STUDIES IN BLOOD-PRESSURE

GEORGE OLIVER

THIRD EDITION

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STUDIES IN BLOOD-PRESSURE

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STUDIES
IN
BLOOD-PRESSURE

PHYSIOLOGICAL
AND CLINICAL

BY
GEORGE OLIVER
M.D. Lond., F.R.C.P.

Edited by W. D. HALLIBURTON, M.D., F.R.S.

THIRD EDITION

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PREFACE

THE untimely death of Dr. George Oliver prevented him from carrying to completion the present edition of his book. He left the major part of it in a condition of readiness for the printer, and this portion was entirely rewritten. His notes indicate that he proposed to write some more chapters, but no manuscript has been discovered. Among the omitted chapters are those on treatment; so I have ventured to reprint what he wrote on that subject in his last edition (1908); but as I did not feel competent to revise this part, Dr. Alfred Mantle, one of Dr. Oliver's old friends at Harrogate, very kindly consented to look through these chapters, and has brought them up to date, such additions as seemed to him desirable being initialled A. M. He has also inserted an addendum to the chapter on the treatment of high blood-pressure.

What appears in the Appendix is also taken from the last edition, and I have added there a brief account of another instrument, namely the hæmoglobinometer, for which Dr. Oliver's inventive genius is responsible.

A few days before he died, he was speaking to me of the importance of arteriometry, which he feared had fallen into disuse; there is certainly no instrument of the many which Dr. Oliver introduced which so completely demonstrates his ingenuity, resourcefulness, and mechanical acumen. I therefore make no apology for reprinting his chapter on that subject. The only other section which has appeared to me necessary to republish from the last edition is the short one on Venous Pressure. All the rest of the book, that is Chapters I to VII, appears as it left the author's pen, except that a few verbal inaccuracies, the inevitable result of typing manuscript, have been rectified. A few footnotes I have added are initialled W. D. H.

In thus editing the present issue I can only say that the work, undertaken at the request of his widow, has been a true labour of affection and respect.

I have also, by the kind permission of the Editor of the *Lancet*, added by way of introduction the obituary notice of Dr. Oliver which appeared in that journal on January 8, 1916, and the leading article of the same date, which seems to me to put most accurately in a few sentences the present position of sphygmomanometry in the field of practice, and the large share which Dr. Oliver had in making it so useful.

W. D. HALLIBURTON.

King's College, London,
May, 1916.

OBITUARY

GEORGE OLIVER, M.D. Lond., F.R.C.P. Lond.

(From *Lancet*, Jan. 8, 1916.)

We regret to announce the death of Dr. George Oliver, which occurred at his residence at Farnham, Surrey, on December 27, 1915. He was born in April, 1841, being the second son of Mr. W. Oliver, a surgeon, of Middleton-in-Teesdale, Durham. He was educated at Gainford School, and having decided to adopt his father's profession he proceeded to University College, where he had a brilliant academic career which culminated in his taking the gold medal at the M.D. examination of the University of London in 1873. Even in these early days he gave evidence of a truly scientific conception of his work, and he always had a deep admiration for William Sharpey, who was professor of physiology at University College at the time. Subsequent to this he started practice at Redcar; he only stayed there a few years, and in 1875 went to Harrogate, where he speedily developed a large practice, first as a general physician and subsequently as a consultant. He spent altogether thirty-three years at Harrogate, and was recognised as the highest authority there in the special branch of practice associated with that town. During the later portion of this period he used to reside in London or in Sidmouth during the winter, only living at Harrogate for the season; in 1901, however, he acquired a beautiful home in Surrey, where he spent the winter months, and it was not until 1908 that he entirely relinquished his Harrogate work. This freedom from the claims of a busy practice during a long period of each year enabled him to prosecute original research, which he loved; some of this was carried out at the Physiological Laboratory, University College, but a good deal in his private laboratory. He was always clever at any mechanical device, and most ingenious and resourceful in overcoming difficulties.

He wrote extensively not only on the Harrogate waters, but also on urine testing; the fourth edition of his "Bedside Urine Testing" was published in 1889, and his originality here was shown by the invention of testing papers known as "Oliver's test papers." But it was always the blood and its circulation that most attracted his attention, and one instrument after another dealing with this branch of science were brought under the notice of the medical world; his hæmacytometer, hæmoglobinometer, arteriometer, and sphygmomanometer are instances. He will be especially remembered for his work in connection with blood-pressure; he it was among British physicians who chiefly insisted on the importance of estimating blood-pressure in diagnosis, and the instruments of precision he introduced for this purpose will live long to keep his memory green. His book on "Studies in Blood-Pressure" rapidly passed through two editions, and at the time of his death he was nearing the completion of a third edition, in which the book was practically re-written. It is to be hoped that this work may ultimately be published.

Another branch of medical science to which he devoted himself was that relating to the ductless glands, and he published papers on the suprarenal, thyroid, thymus, and pituitary bodies. In this direction he was perhaps best known by the remarkable paper, published in conjunction with Professor Sir Edward Schäfer, on the suprarenal gland. This really formed the foundation for all the enormous amount of subsequent work on the adrenal body which has resulted not only in the discovery of the functions of this formerly mysterious organ, but also in the discovery of the active principle adrenaline, one of the greatest boons to suffering humanity. Although Addison many years ago had dimly conjectured the use of the suprarenal capsule, it was not until Oliver and Schäfer published their famous paper that any clear light was thrown upon the subject; and Dr. Oliver at once grasped the practical application of the discovery.

In 1887 Dr. Oliver was elected a Fellow of the Royal College of Physicians of London, and a few years later he founded there, in memory of his old master Sharpey, the lectureship which now bears the name of the Oliver-Sharpey lectureship. Dr. Oliver was most suitably chosen as the first lecturer under this trust, when he took as his subject " Studies on Tissue Lymph " (1904). He had previously filled the position of Croonian lecturer at the same College (1896), which gave him the opportunity of bringing prominently before the profession the importance of a study of blood-pressure. This does not by any means exhaust the list of his writings, for he contributed largely to the journals and also to standard works, such as " Quain's Dictionary of Medicine," in which he wrote articles on constipation and on other ailments of the bowels. He was thus a man with wide attainments. His recreations included photography, in which he became an expert, and until the last few years he took his exercise mostly in the form of cycling. He was never a man of strong physique, but in spite of this he continued to live an active and strenuous life. About five years ago he had an attack of hæmatemesis, from which he fortunately quickly recovered. For about a month before he died he suffered from some bronchial catarrh, and the coughing so occasioned evidently strained the weak spot in his gastric lining. On Christmas night he was again seized with bleeding, which no remedy could check, and two days later he peacefully passed away.

His great aim throughout life was to make medicine a more exact science than he found it, and none can deny that he succeeded in this great endeavour. The keynote of his success, both at the bedside and in the laboratory, was untiring perseverance; he was never satisfied with anything that was imperfect, but went steadily on, altering and improving his inventions until they gave absolutely correct results. It was the same with his writings; he wrote and rewrote each sentence until it was absolutely clear so as to express his meaning in the fewest possible words. He never hesitated to change his views when necessary, and much of what he wrote in his earlier editions was consequently amended as his apparatus were perfected and gave him results which corrected those obtained earlier with less perfect instruments. He was a man of a singularly sweet, genial, and unselfish disposition, which endeared him alike to his patients and his friends. He will be greatly missed not only in the scientific world but also by the large circle of his personal friends in Yorkshire, London, and Surrey.

During the time he was at Redcar he married Alice, the only daughter of Mr. J. Hunt, of Barnard Castle, by whom he had one son and one daughter who survive him. His first wife died in 1898. In 1900 he married again, Mary, daughter of Mr. W. Ledgard of Roundhay, Leeds, and she survives him. Dr. Oliver was buried in the churchyard of the peaceful little church at Tilford on December 31, the church which he used faithfully to attend.

W. D. H.

THE MEASUREMENT OF BLOOD-PRESSURE

(From *Lancet*, Jan. 8, 1916.)

THE progress of science has always been intimately connected with the introduction of instruments of precision. The name of Dr. George Oliver, whose life-work we record in another column, will always be associated with instruments for the measurement of blood-pressure. For although others were earlier in the field in introducing methods capable of clinical application, he was one of the first to make that application and to facilitate it by the simplification and improvement of the apparatus required. And a rich harvest of facts soon rewarded him, even though the interpretation of these facts is still to some extent in dispute. The clinical value of observations on blood-pressure is doubted only by those who have never made them. It is true that the finger can detect some differences in pulse tension, but it is often entirely at fault, since it can only estimate total pressure, thus missing a high pressure if the volume be small. In the same way, although the hand can detect differences in temperature, it cannot replace the thermometer. The sphygmomanometer has enlarged our ideas, cleared up difficulties in diagnosis, and helped in prognosis. This has naturally reacted on treatment. It has enlarged our ideas on "heart failure." The heart is adjusted to work at certain pressures, and though within considerable limits it can adjust itself to variations, those limits may be exceeded. In an acute infection blood-pressure may fall dangerously low, and sensory stimuli do not produce the usual pressor result, though abdominal massage is still effective. It is the vaso-motor system that is poisoned, rather than the heart that is damaged. But the heart cannot carry on the circulation satisfactorily if the blood-pressure is quite inadequate. Recognition of these facts has led to the problem being attacked through the vasomotor system by means which act peripherally, thus protecting the heart from secondary damage and needless or harmful stimulation. Crile and Lockhart-Mummery take a similar view of the origin of surgical shock, though of late this has been disputed. There is the opposite condition in which the heart fails while the pressure is very high. In some cases where the pressure has been unduly high, perhaps for years, the heart fails because it can no longer work against an excessive resistance. But in others it is probable that the rise of pressure is compensatory. With diminishing output vaso-constriction is necessary to lessen the size of the arterial bed. But this vaso-constriction would only raise the blood pressure to its normal level again; it would not force it higher. Yet it is often a surprise to find with a feebly beating heart a pressure of 200 mm. or more. The recent work of Starling and his school throws fresh light on this apparent paradox. The contractile force of a muscle depends upon and is proportional to its previous stretching, so that in its inception even cardiac dilatation is compensatory, and a rise of blood-pressure will increase the stretching of the cardiac muscle. But it is a desperate expedient, for with overstretching complete failure may ensue. In a primary affection of the heart muscle this is particularly likely to occur; hence the liability to sudden and fatal syncope. This work has also thrown a new light on "back pressure," for a rise of venous pressure increases the filling and therefore the stretching of the right auricle. Oliver was much interested in the measurement of venous pressure in man, and published a paper on the subject in 1898, long before its significance was grasped.

This new work explains what had previously been realised clinically—that the indiscriminate lowering of blood-pressure by a frontal attack with vaso-dilators is dangerous. In the early days of the sphygmomanometer there was too great a tendency to its use, just as there was a tendency to indiscriminate lowering of the temperature in the early days of antipyretics. A rise of pressure is a symptom just as surely as a rise of temperature. In all attempts at repair, however, the body may overshoot the mark, and it may become as imperative to lower pressure as to treat hyperpyrexia. The sphygmomanometer has aided in diagnosis in many ways. We may appeal to it if in doubt whether a hemiplegia is due to hæmorrhage or thrombosis; in the former it is always high, while in the latter it need not be, and indeed seldom is. Perforation in typhoid fever causes a rise of pressure, while hæmorrhage produces a fall. A fall of blood-pressure is occasionally a valuable sign of incipient or recrudescent phthisis. A great fall of blood-pressure may occur quite early in Addison's disease. A marked fluctuation on change of posture in a patient with albuminuria suggests that it may be orthostatic in origin. A distinct difference of pressures in the two arms may be a sign of aortic aneurysm. As to prognosis, a continued rise in the already high pressure in cerebral hæmorrhage is of the gravest import. In chronic nephritis a raised pressure is necessary for adequate excretion, and cases in which it does not occur usually do badly. In pneumonia a continued but gradual fall of pressure is the rule. Gibson found that any sudden rise before the crisis implied the onset of some complication, while a sudden fall was a warning of impending vasomotor paralysis, leading to heart failure. A rise of blood-pressure in pregnancy has been found a valuable prognostic sign of the approach of toxæmia. But perhaps one of the most practical applications of the sphygmomanometer has been the recognition of the rise of blood-pressure in later life, before any organic change has occurred in the arteries, and therefore while the case is still amenable to treatment. A busy man nearing fifty years of age, who finds himself getting unduly short of breath on slight exertion, and liable to vertigo, insomnia, "biliousness," or headache, may show no evidence of disease to ordinary physical examination, but the sphygmomanometer would probably reveal a blood pressure of 180 mm. or more. If we meet this by putting the patient into the way of physiological righteousness rather than by depressor remedies, the results are often extremely gratifying.

In this " pre-sclerotic " stage we should pay more attention, as Osler urges, to the peripheral field of the circulation " rather than tinker at the pump or the mains." And the peripheral circulation always received careful consideration in Oliver's work.

It is an interesting paradox that in medicine instruments of precision have a way, largely because of their educational influence, of diminishing the necessity for their own use. Thus the sphygmograph taught us many things about the pulse which could subsequently be recognised without its aid. The electrocardiograph was necessary to the detection of auricular fibrillation and to the explanation of its nature. But already we can sometimes diagnose this condition without it. To a certain extent the same is becoming true of the sphygmomanometer, but this does not diminish our indebtedness to the man who has familiarised us with its lessons.

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STUDIES IN BLOOD-PRESSURE

CHAPTER I

APPARATUS [1]

I. The Manometer

Clinical manometers may be divided into two classes. First, the various forms of the ordinary mercurial manometer measuring pressure by the height of a column of mercury, and secondly, the different kinds of manometers graduated from that standard.

The Standard Mercurial Manometer.—There is a consensus of opinion among the best observers in favour of the mercurial, as the only trustworthy form of clinical manometer. Unfortunately, however, it has hitherto been so handicapped by its cumbrousness and general unsuitability for bedside observation, that the portable instruments

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[1] The apparatus is made by Messrs. Hawksley & Sons, 357, Oxford Street, London.

(aneroids, etc.) graded from it have found favour with many busy general practitioners, though aware of their liability to serious error from the weakening of mechanism contingent on use. But now these objections can be met, as in the instrument I am about to describe. There is, however, one source of fallacy in the mercurial manometer —namely the inequality of the bore, which may produce an error of from 3 to 5 or 6 mm. at least.[1] The bore should be carefully calibrated throughout; and this is a standing instruction to my instrument maker.

A Pocket Mercurial Manometer (unspillable).—Fig. 1 is a small, compact, unspillable, mercurial manometer, which is quite easily carried without indicating its presence in the inside breast pocket. It measures only 9½ inches in length, 1½ inch in breadth, and ⅞ inch in thickness. This little instrument consists of two parts: the manometer and the cover or box in which it is carried; the latter when removed serves as a stand for the manometer; it consists of two layers of wood, and it is detached by releasing the brass spring catch at the upper end of the instrument. Then after sliding the upper piece of wood downwards so as to free it from the dovetailing below it is removed; the central swivelled portion is now turned crosswise, and the cross thus formed is placed upon the table; the brass-covered lower end of the manometer is inserted firmly into the socket at the centre of the cross-shaped stand, and we have an upright manometer firmly fixed for observation (Fig. 2).

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[1] From the following statement by Percival Nicholson (*Blood-Pressure in General Practice*, p. 22) it would seem that American mercurial manometers yield larger errors from imperfect calibration than English ones: "Two instruments recently examined were found to vary 40 mm."

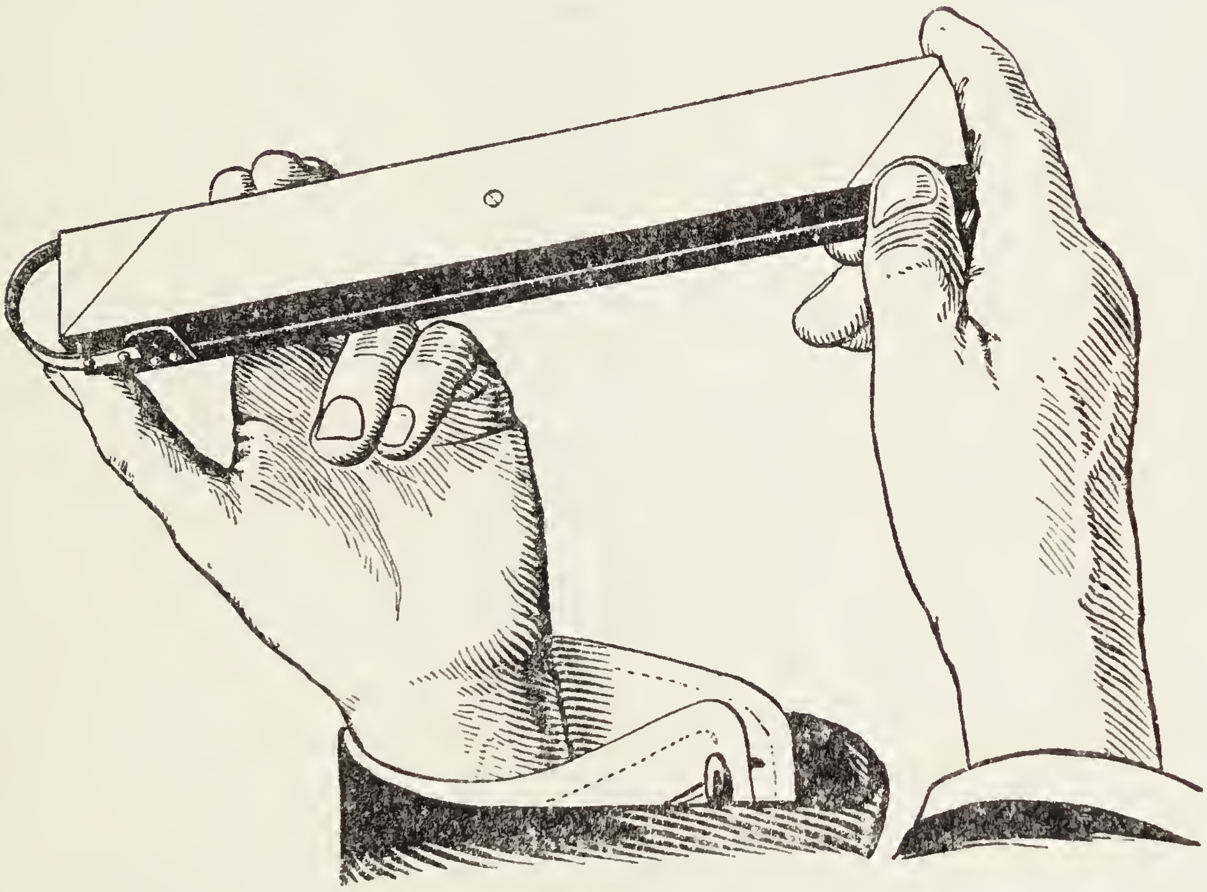

Fig. 1.

By kind permission of the Editor of "The Practitioner."

The manometer differs from the ordinary "U"-shaped instrument in having a separate scale allotted to each limb. The scale affixed to the left limb, reading to 200 mm. Hg., suffices for the general run of cases; and that on the right limb, reading from 200 to 300 mm. Hg., provides for the exceptional cases. The employment of one or the other of these scales is determined by the direction of the arrow on the brass disc. When this is turned home well to the right, the arrow points to the scale on the right limb; but when

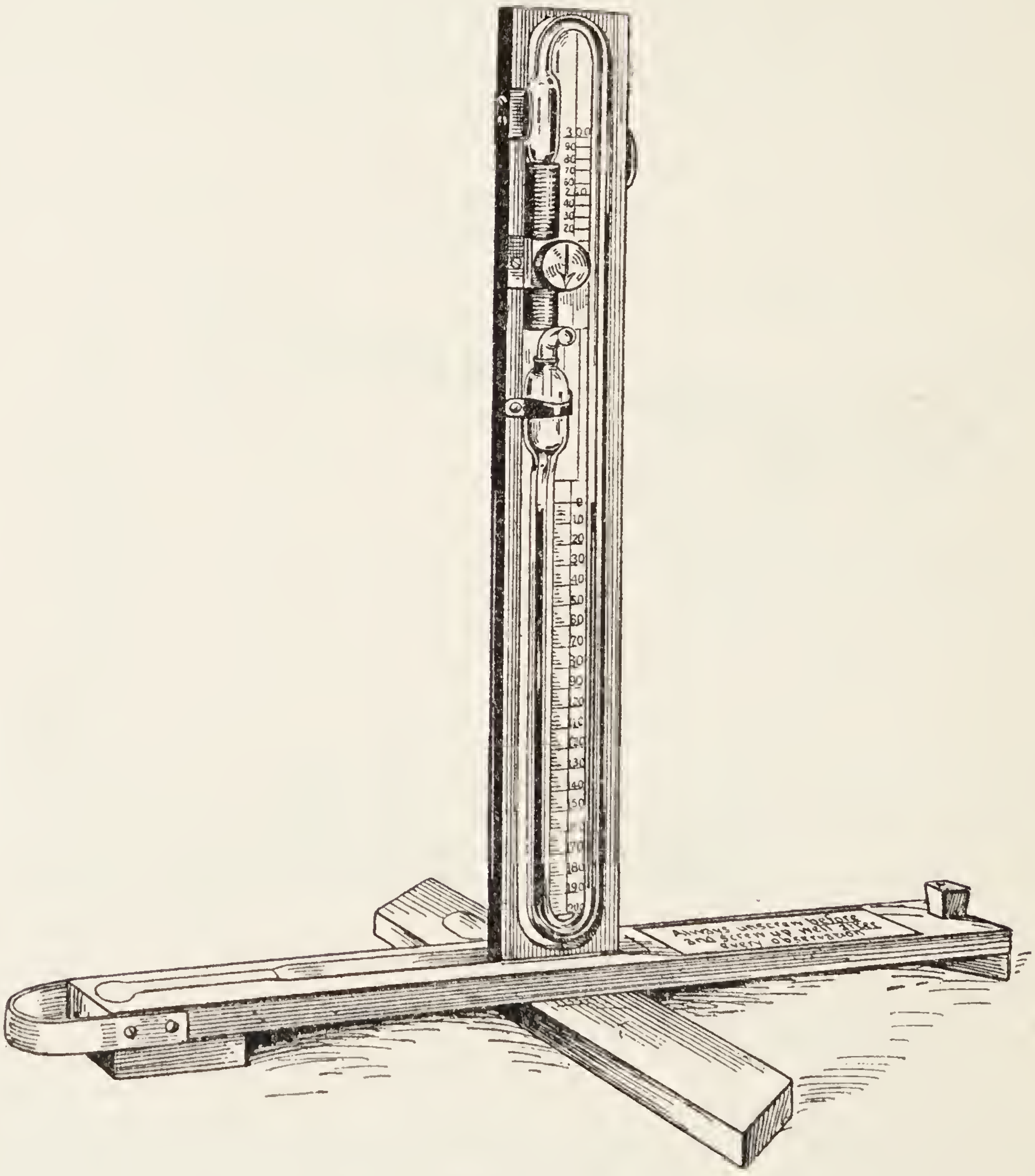

Fig. 2.

By kind permission of the Editor of "The Practitioner."

to the left, until the arrow is directed downwards, it points to the scale on the left limb.

When the brass disc is turned home to the right, the air beyond the mercury is *closed* ; and when to the left, the mercury is *free* to measure the arterial blood-pressure in the ordinary way at atmospheric pressure. This arrangement of supplementing an ordinary manometer reading to 200 mm. Hg., enables us, by closure, to extend the scale to 300 mm. Hg., without lengthening the instrument. In fact, 4 inches in length are thus saved.

The manometer tube is made with a bore of either 2 or 3 mm. in diameter, as the observer prefers. The smaller calibre is just sufficient to damp down oscillation without impairing accuracy : it therefore practically converts the oscillatory into a quiescent column of mercury, and the latter is more suitable for the auditory reading of the pressure than the former, for with the still column the phases of throb are better defined, and the reading of pressure is more exact.[1] But for those observers who, from impaired hearing, are debarred from the use of the auditory method, the larger calibre is provided, which enables them to read the diastolic pressure by the oscillatory, and the systolic by the tactile method. The pressure is read first of all, after turning the brass disc to the left until the arrow points downwards, on the open-air scale below on the left; and should it exceed the limit of that scale, 200 mm., the air in the armlet is released. Then, after seeing that the mercurial column is at zero, the brass disc is screwed up firmly, and the pressure from 200 mm. and onwards is read on the scale above on the right.

---

[1] When the bore is 2 mm., the instrument may require a tilt to set the mercury exactly to zero before making an observation ; but the column of mercury is much less liable to disturbance from shaking and carriage than in the 3 mm. bore.

The observer should never forget to unscrew the brass disc before, and to screw it up after, an observation. The instrument is best carried in the breast pocket; but it may be placed in a bag. In both cases, however, the loop end should always be uppermost.[1] The mercury is trapped at both ends.[2]

---

[1] Should some of the mercury, from faulty position of the instrument, become displaced into the upper trap, it can be returned to the glass tube by (1) holding the manometer upside-down, (2) unscrewing the brass disc, (3) attaching one end of the connecting rubber tube to the nozzle, and then sucking the other until all the mercury is drawn round the bend of the glass tube (the side branch of the connecting tube should be closed by the ebonite plug provided, whilst this is being done), when the instrument is quickly turned the right way up. Any detached pieces of mercury are easily united by shaking. Such an accident as this is rare, and should not happen if care be taken to follow the directions for the use of the instrument.

[2] Those manometers which are trapped by a glass arrangement

A Pocket Compressed Air Mercurial Manometer (unspillable).—The reading of the arterial pressure by means of compressed air throughout (as described in the second edition of this work) possesses many advantages. But it is open to one disadvantage, namely the influence of barometric pressure: but this is counterbalanced by the elimination of error from imperfect calibration of the bore of the tube, for the instrument is graduated from a standard manometer. Some observations, made with the view of ascertaining the extent of error due to altitude, led me to infer that 1,000 ft. add about 5 per cent. to the graduations on the scale which is set up within 100 ft. above sea-level; so that 100 mm. at that altitude should be read as 95, 200 mm. as 190, etc. From these figures we may conclude that the error is practically negligible for districts under 500 ft. in height, and may be approximately calculated for higher levels. Barometric rises and falls of half an inch (practically equivalent to 500 ft.), which are not common, may also be disregarded. My compressed air manometer, in an improved

at both ends cannot be cleansed from deposits of mercurous oxide; they must therefore be discarded when dirty. But in this manometer, the lower trap is removable, and consequently the bore can be cleaned and refilled with doubly distilled mercury by the instrument maker at a nominal cost.

form, can be procured by any one preferring to use it.

II. The Armlet

The armlet is of standard width (12 cm.), and is furnished with an air-bag of limited area (12 × 12 cm.). The observer has the choice of two forms; one with three automatic catch buckles and a central tube, and the other shaped as a bandage with a marginal tube, and two straps punched with eyelet holes which hook and unhook with facility. The latter form of armlet is very convenient for applying the pressure of the hand over the armlet when the observer wishes to avoid constriction of the limb in sensitive subjects.

For differential readings of the arterial pressure in the two arms, or forearms, or in the arm and thigh (in aortic insufficiency, etc.), two armlets are provided, furnished with a tap for each, and rubber tubes with a T-way piece; the one adapted to the thigh is larger, and of suitable dimensions.

III. The Canvas Bag

A small short canvas bag (8 × 10 cm.) is employed to ensure quick compression and decompression of the armlet. It is connected up between the filler and the tubes leading to the armlet and the manometer. The filler is furnished with a tap as well as the valve inside, so that should the latter become leaky, the observer is independent of this defect.

IV. The Phonendoscope

A sensitive phonendoscope (Fig. 3) is provided for the efficient application of the auditory method.

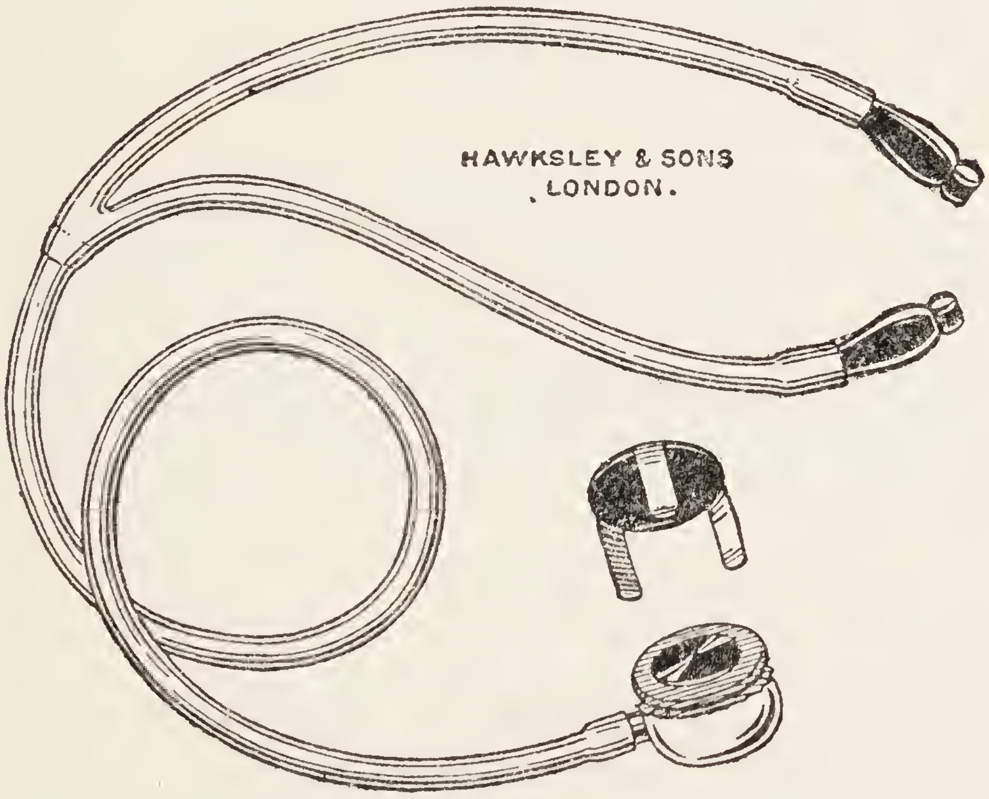

Fig. 3.

It is disc-shaped, of less than 3 cm. in diameter, and is fitted with a binaural rubber tube in one piece, with ear plugs. For those who prefer a firmer fixture in the meatus than the plugs alone afford, a head spring is made.

When devising this little phonendoscope, I discovered that its maximum degree of sensitiveness depends on absolute air-tightness: a condition not found in other phonendoscopes. This important quality is lost even by the invisible prick of a needle passed through the rubber. The observer may easily test the air-tightness of the instrument by suction of the entrance tube, after withdrawing from it the rubber tube, and then closing it with the tip of the tongue, when the rubber cover will remain concave if the tambour is air-tight; but will not do so if the tambour requires recovering.[1]

A stout rubber band, adjustable to all sizes of arms, is provided for keeping the phonendoscope *in situ* without pressure just below the armlet.

The advantages of the phonendoscope over the end-piece of a stethoscope are: (1) increased sensitiveness; (2) avoidance of pressure, and especially of variable and uneven pressure over the artery; and (3) liberation of the finger of the left hand for palpation of the pulse, a necessary adjunct to the auditory method. A further advantage is that the phonendoscope can be efficiently used for auscultation of the heart; when thus employed it should be applied to the chest, under the gentlest pressure—the ebonite disc attached to the rubber cover merely touching.

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[1] Messrs. Hawksley will recover it at a nominal cost.

V. The Screw-Compressor

Many observers have found the screw-compressor (Fig. 4) more satisfactory than the ball or pump filler in the consulting-room.[1] It consists of (1) a strong canvas bag adherent to (2) two small boards (12 × 18 cm.), through which (3) a strong rapid screw passes. When in use, the tap (A) is opened for the admission of air; the wheel (B) is unscrewed to the limit of the screw; tap A is then closed, and the wings of the screw are turned, when air is compressed into the air-bag and manometer, previously connected by the rubber tubes (C and D).[2]

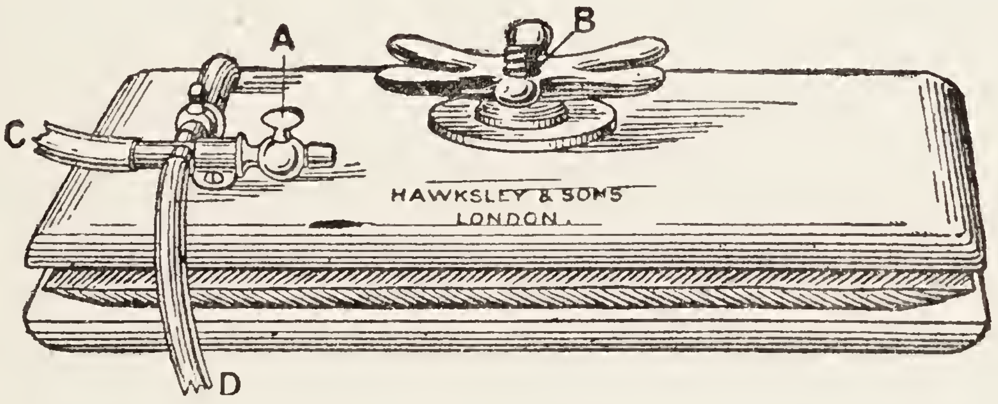

Fig. 4.

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[1] J. A. MacWilliam and G. Spencer Melvin thus refer to it in *Heart*, vol. v, p. 192: "For conveniently graduating the application of the external pressure, Oliver's screw-compressor, when employed with a suitable armlet (with a rubber bag of not too large capacity), is very useful."

[2] The air capacity of the compressor is so adjusted to that of the armlet as to secure compression to 300 mm. when the armlet is closely adjusted. Should the easy turning of the screw of the

The practical advantages of the compressor are:

1. The avoidance of leakage, and its consequence—a falling index—from a defective air-valve, which cannot be rectified without loss of time and trouble. This not infrequent defect of the ball or pump filler, always a source of annoyance and inaccuracy in observation, is entirely obviated by this valveless arrangement.

2. The much more satisfactory control of the mercurial index by the screw than by ball or pump filling. The adoption of screw-compression is definite and easy.

3. The smooth and even rise of compression, which may be made as rapid or as slow as the observer desires, in place of the intermittent increments of it with every stroke of the ball or pump.

4. Increased precision in reading the pressures (systolic and diastolic)—the least turn of the screw deciding the exact point at which it should be made.

It is true that these advantages are practically secured by the ball filler supplemented by the canvas bag; but in the consulting-room the screw-compressor conveniently combines both these parts of the apparatus.

compressor fail, it will be restored by letting a drop of oil fall on the screw when the wheel is unturned to its fullest extent.

CHAPTER II

FUNDAMENTAL DATA

There are certain physiological data which the observer in his preliminary study of the arterial blood-pressure should clearly grasp. I shall briefly discuss the leading ones which underlie the clinical variations of the pressure; and in doing so I shall endeavour as much as possible to trace from the basis of physiology the beginnings of pathological deviations which find expression in abnormal arterial pressure.

I. The Heart

Though the left ventricle—dispensing its load under a high pressure into the aorta—is the master hand in maintaining the arterial blood-pressure, the height to which that pressure rises depends primarily on the resistance encountered in the peripheral arterial system, and in the capillaries, and secondly on the response of the ventricle to overcome that resistance. Given a normal ventricle, the arterial tonus largely determines the ventricular response, and the level of the arterial blood-pressure; and this is the keynote of the physiological and pathological variations of that pressure.

Weight of the Heart as Life Advances.—The heart continues to increase in all its dimensions as life advances, viz. in length, breadth, and thickness of its walls, the greatest increment being detected in the substance of the left ventricle and the ventricular septum (M. Bigot)[1]; and with every advancing decade the weight of the organ rises (J. Reid,[2] T. B. Peacock,[3] J. Glendinning,[4] and Wilh. Müller[5]). J. Reid's figures also indicate that in males the decades 40–50 and 60–70, and in females 30–40 and 50–60, furnish the largest increments of weight; and that in women after 60, the weight slightly decreases: whereas in men it continues to increase markedly. These facts are in conformity with the effect of work on the heart muscle during the course of life, and especially with that due to the increased peripheral resistance which normally sets in after the middle period. There is a slight decrease in weight during the years of senile involution (70–100),[1] when the work of life declines generally.[2]

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[1] *Mém. de la Soc. Médic. d'observation de Paris*, tom. i, p. 262.

[2] *Lond. and Edin. Monthly Journ. of Med. Science*, 1843, p. 323.

[3] *Ibid.* 1846: "On the weight and dimensions of the heart in health and disease."

[4] *Med. Chir. Trans.*, 1838, vol. xxi, p. 33.

[5] *Die Massenverhältnisse des menschlichen Herzens*, 1883, p. 57.

The Heart as a Mechanism.—E. H. Starling and his co-workers (F. P. Knowlton, J. Markwalder, S. W. Patterson, and H. Piper) at the Institute of Physiology, University College, London, have opened up an interesting chapter on the mechanical behaviour of the mammalian heart, isolated from all nervous control, but with intact respiration, under known conditions of arterial resistance (arterial blood-pressure), inflow and rate. This is technically known as "the heart-lung preparation." The following are the leading results of this investigation:[3]

1. When the inflow (venous filling) is maintained constant, the output from the left ven-

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[1] Geist in *Klinik du Greisenkrankheiten*, 1860.

[2] Lewis has observed (*Heart*, vol. v, 1914, p. 367) that much of the substance of the human heart is non-muscular, whereas in dogs it is chiefly muscular. This fact somewhat reduces the cogency of the recorded figures of the weight of the heart in the successive decades of life, if we regard them as representing a purely muscular organ; at the same time I do not think that it invalidates the teaching which these figures afford.

[3] *Journal of Physiology*, vol. xliv, 1912, p. 206; vol. xlvii, 1913, p. 275; vol. xlviii, 1914, pp. 348, 357, 465.

tricle[1] is unaltered by rate of heart-beat or by the resistance presented by arterial pressure. The mechanism is therefore perfect for the complete systemic delivery of all the blood supplied to the heart by the veins.

2. Within wide limits the heart is able to increase its output in direct proportion to the inflow. In other words the arterial output of the heart is controlled by the venous input. Therefore under normal conditions the greater the arterial resistance the higher will be the venous pressure for any given inflow, and the sooner will the inflow attain its optimum and the output its maximum.

3. The coronary circulation is intimately connected with the arterial blood-pressure, rising and falling with the pressure at the root of the aorta ; and is independent of the total cardiac output. It is, moreover, specially increased by non-volatile metabolites generated by the heart muscle, these products being the most potent agents in causing dilatation of the coronary vessels.[2] The action of the heart muscle, therefore, provides a local

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[1] The ventricular output is the volume of blood per heart-beat measured beyond the opening of the coronary vessels *plus* that which circulates through the walls of the heart.

[2] Adrenaline, in small as well as in large doses, and increased tension of carbon dioxide in the blood also dilate the coronary vessels (*Journal of Physiology*, vol. xlvii, 1913, pp. 281–2).

mechanism by which the circulation within itself is increased to meet the calls made on its functional capacity; and thus the metabolism of the heart increases or diminishes in proportion to the mechanical demands made upon it, the chemical sources of energy being automatically drawn upon in proportion to its requirements.

4. Increased arterial resistance increases the volume of the ventricle and prolongs the duration of the systole, and shortens that of the diastole. As already mentioned, it likewise increases the venous inflow; though this influence is limited by certain degrees of resistance in the arterioles and small arteries.

These observers remark that "it is difficult to imagine a more perfectly regulated machine than the heart"; and they infer that the underlying agency of this remarkable self-regulating capacity depends on the length of the muscular fibres when in a state of tension, *i.e.* during contraction. It is a well-attested fact in physiology that when a muscle is stretched to, or beyond, its physiological length, or the maximum length ever assumed during life, its mechanical energy set free in contraction is increased;[1] the muscle being then converted into a new state possessing changed elastic properties which augment the tension energy expressed either in work or heat.[1] The experimental data show that the heart reacts to alterations in the arterial pressure or resistance by changing its mean volume; and adapts itself to a rising pressure by dilating until its muscular fibres assume such a length that on their contraction they produce sufficient tension to overcome the increased resistance, and then it continues to empty itself as effectually as before.[2]

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[1] *Textbook of Physiology*, edited by Sir E. Schäfer, F.R.S., vol. ii, p. 243.

The Regulating or Control Mechanism of the Heart.—The rate and rhythm of the contractions of the heart muscle are controlled by nerve structures. The mechanism for this purpose consists of the nodes and bundles of neuro-muscular fibres situated in the heart wall, and of the nerves supplied to the heart. The former must be regarded as the fundamental or intrinsic part of the cardiac regulatory system (a fact which is proved by experimental work on the heart-lung preparation) which initiates the contractions of the cardiac musculature and sets their rate; whereas the latter, or extrinsic portion, are the channels (vagus and sympathetic) and nerve cells or ganglia scattered over the heart through which nerve impulses derived from all parts of the body inhibit or accelerate the heart's action.

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[1] Weber in *Wagner's Handwörterbuch d. Physiol.*, 1846, Bd. iii, Abth. 2, S. 110; A. V. Hill in *Journ. of Physiol.*, vol. xlvi, 1913.

[2] It was, however, observed that for six or eight beats after raising the arterial resistance the heart failed to empty itself completely.

The slowing of the heart by compressing both eyeballs by the fingers or of the brain by a clot or tumour, etc., or a blow on the stomach, and the quickening of it by fever or nervous disturbance are examples of this reflex linking-up of nervous impulses derived from various organs and tissues with the neuro-muscular cardiac mechanism.

Certain portions of the neuro-muscular tissue[1] in the heart wall originate contraction and determine its rate. Two such centres are situated in the right auricle; one the sino-auricular (S.A.) node, between the mouths of the venæ cavæ, and the other the auriculo-ventricular (A.V.) node, in the posterior part of the right auricle. These nodes are connected in the auricular septum; and from the A.V. node there proceeds a bundle of neuro-muscular fibres (the auriculo-ventricular bundle) which is distributed along the intra-ventricular septum to the internal portions of the heart wall as far as the apex and terminates in the musculi papillares. The S.A. node has been denominated "the pacemaker of the heart"; and the A.V. node co-ordinates the auriculo-ventricular contractions. Provision is thus made for the co-ordinated contraction of all the chambers of the heart mechanism.

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[1] The tissue in these portions, and in the auriculo-ventricular bundle, consists of those primitive muscular fibres known as the fibres of Purkinje; although nerves are present, it is generally admitted that the functions of initiating the beat and conducting the impulse belong to the muscular rather than to the nervous tissue present. (W. D. H.)

This system of fibres, when disorganised by fatty degeneration, is the seat of the more serious form of the pathological irregularities of the heart. Several observers,[1] for example, have proved that one of these arhythmias, heart-block, is traceable to interruptions in the A.V. bundle; for when in animals this bundle is clamped, crushed, or cut, heart-block, either partial or complete, such as is met with in clinical observation, is produced—the normally co-ordinated auricular and ventricular rhythms becoming dissociated and independent, and the latter always slower than the former.

When the pulse is irregular, as in heart-block, auricular fibrillation or flutter, extrasystole, or in "pulsus alternans," the ventricular contractions are arhythmic not only in point of time, but in that of force. Hence it is that irregularity of the pulse makes it somewhat difficult to estimate the arterial blood-pressure, as this varies in the individual pulsations, or in many of them, and the observer can only express it in terms of minimum and maximum limits. Some forms of irregularity, however, interfere with observation of the pressure in a much less degree than those due to "heart-block." For example in "extrasystole," when the interruptions are not too frequent, a fairly satisfactory reading can be made; and in "pulsus alternans" the weaker alternate pulsations can be eliminated at a lower level of pressure, the pulse rate being thus reduced to one-half, and the remaining stronger beats can be read off at a higher level.[1]

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[1] Wooldridge, *Ludwigs Arbeiten* and *Arch. f. Anat. u. Physiol.* 1883, p. 533; Tigerstedt, *Ludwigs Arbeiten* and *Arch. f. Anat. u. Physiol.*, 1884, p. 503; MacWilliam, *Journ. of Physiol.*, 1886, vol. ix, p. 177; Joseph Erlanger, *Journ. of Exper. Med.*, New York, 1906, vol. viii, p. 13. Cited by Sir Lauder Brunton, *Therap. of the Circulation*, 2nd ed., 1914.

Cardiac irregularities and the time relations of the auricular and ventricular portions of the rhythm of the heart muscle have been investigated with a rich harvest of results by Sir James Mackenzie's polygraph[2] and by the string galvanometer.[1] These instruments reveal the mode of working of the cardiac mechanism; but they are not designed to estimate the blood-pressure in the peripheral vessels, which is the province of the manometer.

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[1] C. O. Hawthorne, "Pulsus Alternans in its Clinical Aspects," *Lancet*, 1915, vol. i, p. 1125.

[2] *Diseases of the Heart*, by Sir James Mackenzie, M.D., F.R.S., etc., 1908.

The Rate of Systole.— When the rate is increased or diminished, the normal duration of diastole is disturbed to a far greater degree than that of systole. For example, when the heart's action is quickened by stimulating the accelerator nerve in animals, the length of the diastolic or resting interval is shortened (*e.g.* from 0·299 to 0·044 sec.) far more than that of the systolic period (*e.g.* from 0·253 to 0·210 sec.); and when it is slowed by excitation of the vagus the diastolic rest is more prolonged (*e.g.* from 0·373 to 0·635 sec.) than during systolic activity (*e.g.* from 0·149 to 0·158 sec.).[2]

As it is generally held that prolongation of diastolic rest within certain physiological limits is favourable to recuperation of the heart muscle, it is not improbable that an abbreviation of the heart's resting period by acceleration of the rate of systole beyond that normal to age will tend to impair the well-being and integrity of the heart as a mechanism. But in drawing this inference we should keep in mind other qualifying conditions, such as a rise in the systolic pressure, which prolongs the duration of the systole and increases the volume of the coronary supply (p. 16). Is it not probable that the rise of systolic pressure observed in the later decades of life is an important factor in maintaining on a higher level the metabolism of the heart muscle?

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[1] A. Waller, *Journ. of Physiology*, 1887, vol. viii, p. 229; *Lancet*, 1913, vol. i, p. 1435; Einthoven, *Annal. d. Physiol.*, 1903, vol. xii, p. 1059; Lewis, *Mechanism of the Heart's Beat*, 1911; and other workers too numerous to mention. The reader will find an excellent résumé of the construction and use of the string galvanometer in Sir Lauder Brunton's excellent little manual, *Therapeutics of the Circulation*, 1914.

[2] *Textbook of Physiology*, by Sir A. E. Schäfer, F.R.S., etc., vol. ii, p. 39.

But augmentation of the systolic pressure does not seem to be a necessary condition of efficient metabolism throughout life. In the constructive or anabolic form, which predominates from birth to the completion of adolescence, the pressure is much lower and the frequency of ventricular contraction is greater than in the middle or in the later periods of life. It would seem that in the growing stage the anabolic activity of the cell is at its optimum and merely requires an ample supply of blood under a comparatively low pressure; but as life advances into the settled periods in which the katabolic side of metabolism develops more fully, the aid of increased pressure seems to be more and more needful.

Experiments on the heart of the frog or mammal show that the rate of systole also influences the arterial pressure; for when it is reduced by stimulating the vagus the pressure falls, and when it is increased by cutting both vagi or by stimulating the accelerator nerve the force of the contractions increases.

The Total Energy or Mechanical Efficiency of the Mammalian Heart has been estimated in the "heart-lung preparation" by the oxygen usage or gaseous metabolism of the heart (Evans and Starling,[1] Evans and Matsuoka[2]). Evans and Matsuoka have observed that the total cardiac energy indicated by oxygen usage during exercise is at least three times that recorded during rest; that other conditions of experiment being the same, it is increased by raising the arterial blood-pressure, by augmenting the output, and in a smaller degree by accelerating the velocity of the blood-flow; and that whenever it is thus raised the increment advances to a certain limit beyond which it diminishes again. Fatigue phenomena were seen in a lowered efficiency accompanying cardiac dilatation. These results accord with the close parallel between cardiac energy and heart volume (length of the cardiac fibres) demonstrated by Patterson, Piper, and Starling.

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[1] *Journ. of Physiology*, vol. xlix, 1914, p. 67.

[2] *Ibid.* vol. xlix, 1915, pp. 378–405.

The aggregate work of the human heart in twenty-four hours can only be expressed in huge figures. G. N. Stewart calculates that the normal daily work of the two ventricles is about 22,000 kilogramme-metres, which is enough to raise a weight of 6 lb. from the bottom of the deepest mine in the world to the top of the highest mountain, or to raise the man himself to twice the height of the spire of Strasburg Cathedral, or three times the height of the loftiest "sky-scraper" of New York.[1] These illustrations of the twenty-four hours' work of the normal heart, maintaining a systolic arterial pressure of 120 mm. Hg. or so, are striking enough; but a calculation based on the work of a ventricle sustaining an arterial pressure of from 200 to 240 mm. Hg. must yield still more impressive figures of the enormous contractile energy of the heart expended day by day.

Augustus Waller estimates the duration of the daily work of the heart as nine hours, the resting period being fifteen hours,[1] and he regards a heart working only eight hours as a good one, and a heart which works thirteen hours as unsatisfactory.[2]

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[1] *A Manual of Physiology*, by G. N. Stewart, M.A., D.Sc., etc., 5th ed., 1906, p. 113.

Inefficiency of the Heart Muscle. Increase of Heart Volume. Dilatation.—In the course of their experiments, E. H. Starling and his coworkers observed that the power of adaptation of the heart muscle to variations of inflow and arterial resistance has its limits. They found that the musculature tends to deteriorate steadily during the course of an experiment, as shown by the fact that at the end of systole the heart will contain more and more blood, and consequently its muscle fibres will work under an increasing mechanical disadvantage. This failure may show itself either under the stress of high resistance, or, after reducing the arterial pressure, the heart, after apparent recovery of volume, may slowly dilate under the lowered pressure; and it "can occur either with a small or a large inflow, only the failure will occur at a lower arterial resistance with a large than with a small inflow."[3] Inefficiency of the heart muscle is due to diminution of physiological fitness or tone, which is dependent on the supply of oxygen *plus* nutrient material conveyed by the coronary circulation and of metabolites generated by the contracting muscle itself (see p. 16). Though the coronary flow is proportionate to the arterial pressure, the muscle fibres may, nevertheless, give out under the stress of a high pressure when the inflow is large. Primary dilatation of the heart, which may slowly develop after the sudden lowering of arterial resistance, is due to the diminished blood supply to the heart muscle. Observation of the normal isolated heart therefore shows that under a rising arterial resistance the heart volume is increased whether the musculature is efficient or not; the only difference being that in the former case the inflow is completely discharged, and the diastolic level is therefore unaltered, whereas in the latter residual blood collects in the heart between the contractions and dilatation becomes more and more pronounced.

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[1] *Introduction to Human Physiology*, by Augustus Waller, M.D., F.R.S., p. 52.

[2] Oliver-Sharpey Lectures, *Lancet*, 1913, vol. i, p. 1520.

[3] *Journ. of Physiol.*, vol. xlviii, 1914, p. 484.

Evans and Matsuoka[1] have shown in their experiments on oxygen usage of the heart that fatigue phenomena begin with lowering of the gaseous metabolism and retardation of the pulse rate. These observers also remark "that as the heart becomes exhausted, its volume increases and its gaseous metabolism is increased. Supposing now that the heart ultimately fails, how does its gaseous metabolism behave? The answer is that the heart may fail in one of two ways, and that the gaseous metabolism will be influenced by the mode of failure. The first method of failure is that the heart is confronted with work which is too great for it, it dilates maximally, and ceases to expel its contents, thereby dying of asphyxia; if we reduce the inflow or pressure and empty the heart by massage or by drawing blood from veins before it succumbs, it will resume work. There is every reason to suppose that in such a case the energy usage of the heart is maximal until it is asphyxiated. There is no slowing until asphyxial symptoms set in in the heart tissue. . . . The result of one of our experiments suggests that there is a second method of failure, viz. by exhaustion, in other words by failure of its metabolic processes, which is of course the terminal condition in the other mode of failure if the condition of stress is not relieved sufficiently early."[1]

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[1] *Op. cit.*

These physiological data demonstrate that the primary effect of stress from increased arterial resistance on the heart muscle is to elongate the muscular fibres—in other words, to dilate the heart. In clinical work the same sequence of events is constantly found; for the first effect of hypertension is to dilate the heart, as shown by the moving outwards of the apex beat [1] and by the increased area of the heart demonstrated by percussion and by the auscultatory use of the tuning-fork.

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[1] *Op. cit.*

Compensatory Hypertrophy.—The first step in compensation to meet a rising arterial resistance is stretching of the cardiac muscular fibres; and this, as we have seen, is proved by physiological experiment. But the next step to further enforce contraction so as to prevent loading during diastole cannot be demonstrated in the laboratory, because in an experiment the time required for its development is not available. That step is the proliferation of muscular fibres known as compensatory hypertrophy; and the proof of its existence is amply provided by pathological observation. It has, however, its physiological counterpart, though in a minor degree, in the normal hyperplasia which contributes to the increased weight of the heart in the course of life.[2] The conditions necessary for its development are (1) the persistence of the increased arterial resistance, (2) the amplitude and efficiency of the coronary circulation, and (3) the presence in the blood of a sufficient supply of non-volatile metabolites and adrenaline, oxy-hæmoglobin, and a plasma well charged with constructive elements. Clinical observation, for example, has shown that when the coronary circulation is defective, as in some cases of moderate or slight hypertension with congenital or sclerotic narrowing of the coronary arteries, compensatory hypertrophy may be insufficiently established, and sudden cardiac failure may occur with or without anginoid symptoms.[1]

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[1] *Blood and Blood-Pressure*, by the author, 1901.

[2] Another contributor is the proliferation of fibrous tissue.

The high arterial pressure of compensatory hypertrophy from increased arterial resistance is accounted for by the fact that the hyperplasia is implanted on the dilated state of the heart muscle; for it is well known that the force of contraction is proportionate to the increased radii of the ventricular cavity.[2] In this way the heart becomes a much more powerful organ than is ever met with under ordinary physiological conditions, and is doubtless capable of maintaining efficiently all the higher ranges of systolic blood-pressure met with in clinical observation.

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[1] Guthrie Rankin, *Lancet*, 1914, vol. ii, p. 820.

[2] *Textbook of Physiology*, by Sir A. E. Schäfer, F.R.S., vol. ii, p. 40.

Decompensation.—Compensatory hypertrophy due to increased arterial resistance may maintain the efficiency of the circulation for many years. As a rule, however, it ultimately drifts into decompensation, when the muscular fibres, invaded by fibrotic and fatty degeneration, weaken and become unable to deliver their full load, and the volume of the heart further increases; and the heart's action ceases from cardiac failure.

The transition from competency to incompetency is generally indicated by an alteration of the blood-pressure in the arteries and veins. As a rule the arterial pressure may fall somewhat, though it frequently does not; but the venous pressure undergoes a much more decided change, maintaining a higher level which increases *pari passu* with the incompetence of the heart muscle.

II. THE ARTERIES

In order to realise the part played by the arteries in determining the blood-pressure within them, it is not necessary to study their structure in minute detail. Therefore I trust the following summary will suffice for the purpose of this work.[1]

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[1] In writing this summary I am much indebted to the excellent *Report on Arterial Degeneration and its Premature Occurrence* by F. W. Andrewes, M.D., F.R.C.P., F.R.S. (the Government Report of the Medical Officer, 1911–12, published in 1913).

The Properties of the Elemental Tissues of the Arterial Wall.—Of the three tissues employed in building an artery, the fibrous serves for scaffolding and resistance, the elastic for stretching and recoil, and the muscular for contraction and relaxation under sympathetic nerve control. Speaking broadly, the fibrous tissue is practically inactive, whereas the elastic and muscular are the active or working tissues. They are all liable to degeneration, to automatic hyperplasia in response to requirement, and to be the seat of deposition of material (calcium and cholesterol-fats) conveyed to them by the blood from elsewhere. The nutrition of these tissue-elements is maintained by the "vasa-vasorum" which ramify in the adventitia and the outer third or half of the media, and by the blood which bathes the intima.

The General Distribution of the Tissue-Elements in the Arterial Wall.—The scaffolding of fibrous tissue exists in much the same proportions in all arteries (large and small), preponderating in the outer coat and pervading in an inconspicuous degree the middle coat, where it supports the other tissues, vessels, and nerves. But the dynamic tissues (elastic and muscular) are not distributed throughout the arterial system in anything like the same degree of uniformity.

In the larger arteries (such as the aorta, pulmonary, common carotid, subclavian and common iliac) the proportion of elastic tissue predominates over that found in the smaller vessels. These arteries are therefore classified as "elastic." In the aorta, for example, the thick sheets of elastic tissue disposed throughout the middle coat are equal in bulk to that of the muscular element.

The medium-sized and smaller arteries and arterioles distributed to the different organs and tissues possess a similar preponderance of unstriped muscle, and are therefore denominated "muscular" arteries. In them muscle constitutes practically the whole of the middle coat, the elastic tissue consisting only of a few straggling branching filaments among the muscle fibres, and two layers (internal and external elastic laminæ) between the middle and the outer and inner coats.

The Functions of the Elastic and Muscular Elements.—A good notion of the part played by the elastic and muscular tissues of the arterial wall in maintaining the normal circulation may be realised by conceiving what would happen if the arteries were mere fibrous or rigid tubes; for in that case the frictional resistance to be overcome by the heart would be enormous, and the arterial blood-pressure would alternate between a high maximum and a low minimum degree—the latter approximating to zero in proportion to the volume of outflow into the capillaries. The absence of the elasticity and musculature of the arterial wall would therefore involve waste of cardiac energy and reduction of efficiency throughout the whole circulation.

The elasticity of the arterial wall possesses the rubber-like property of yielding under internal pressure and of recoiling when the pressure is withdrawn; and experiment shows that this capacity is complete, the recovery being absolute on cessation of internal stress.[1] In this way the work of the heart is economised; for the output of the intermittent ventricular contraction stretches the large vessels, and the energy thus stored is expended by their recoil during diastole. Thus it is that, while the heart is recuperating after its effort, the elasticity of the great vessels is the most effective agent in securing a sustained flow through the arterial system at a much higher level of minimum pressure than would be at all possible in fibrous or rigid vessels.

But surely in performing this important work the elastic elements of the great arteries must be enforced by the co-operation of the muscle fibres amply present in their middle coat. Such a conjoint agency is apparently the teaching of experiment which shows that arteries can practically empty themselves into the veins when the heart's beat is stopped by vagus inhibition;[1] for it is difficult to conceive that the elasticity of the arteries alone will suffice to unload them.

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[1] *Textbook of Physiology*, edited by Sir E. A. Schäfer, F.R.S., vol. ii, p. 74.

The musculature of the arteries (1) aids the flow of blood through the arterial system and is thus ancillary to the heart and arterial elasticity; and (2) it regulates, by virtue of its contraction and dilatation under sympathetic nerve stimulation, the distribution of blood to the different organs according to their requirements in states of action and rest. A good example of the regulating capacity of contractility is afforded by the cutaneous circulation.

The arterial wall varies very greatly in thickness and in distensibility to internal pressure in different individuals of the same age and at different ages. In wasting diseases (such as phthisis, etc.) it attenuates and softens, and becomes abnormally distensible, and generally after middle life it gradually stiffens as the years advance, and its distensibility lessens. In the former case the trend of the systolic pressure is downwards, and in the latter it is upwards. In the less distensible artery, the pressure not only rises, but the range of its variations is considerably extended; so that any increment of the ventricular output, of the force of ventricular contraction, of the volume of the blood, and of the peripheral resistance, are more effective in raising it, than when the arteries retain their normal softness and elasticity. The elastic tissue richly distributed through the arterial wall forms a perfect spring which works conjointly with the normal musculature to mature manhood; then, or about that time—it may, in individual cases, be earlier or later—the stress of life-work [1] on the arterial wall induces a proliferation of the fibrous and muscular tissues,[2] which should be regarded as an adjustment for the prolongation of the working capacity of the arteries. When the co-efficient of elasticity begins to fail, it may be that the walls of the large central arteries, which are sparsely endowed with muscular tissue, become braced by some slight proliferation of fibrous tissue[1] in the tunica media and tunica adventitia; and thus the chances of further weakening of the spring under the strain of the work imposed upon it are reduced. But whatever advantage may be thus gained, will be at the expense of limiting the range of the elastic play of the arterial wall; a defect which may be met by the proliferation of the musculature in the tunica media, which is the copartner of elasticity in the earlier years of life, and now becomes the leader. Hyperplasia of the muscular tissue of the artery is the important adjustment when the play of the arterial wall becomes reduced by proliferation of fibrous tissue; for such an artery, without this compensation, would ultimately resemble a rigid tube through which the pressure of fluid intermittently injected falls to zero, or practically to zero, between the strokes. Observation, however, demonstrates that the diastolic pressure in normal elderly subjects with palpable arteries does not fall, and as a rule remains unaltered, or may even be slightly raised ; and this fact can only be accounted for by the proliferation of muscular fibres which holds up the trough of the pulse wave to the normal level or slightly above that level. The compensation by hyperplasia of the musculature may exceed or fall short of physiological requirement, and may either raise somewhat the diastolic pressure or lower it. In the latter case, as in atheroma, the superficial arteries (*e.g.* the temporal, etc.) are apt to become tortuous.[1]

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[1] *Textbook of Physiology*, edited by Sir E. A. Schäfer, F.R.S., vol. ii, p. 74.

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[1] On realising that the aortic wall is stretched by the ventricular output over 1,576 million times in forty years, the pulse rate being taken as seventy-five per minute, we can well conceive how its elasticity may become impaired on the attainment of middle life.

[2] The local proliferation of the musculature in the radial artery so frequently met with in hand labourers is generally the outcome of local physical overwork.

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[1] The development of fibrous tissue in the arterial wall is probably only a part of the generalised hyperplasia of it which pervades the fibro-elastic tissue throughout the body in the elderly and aged. It is not improbable, however, that it may sometimes invade the arteries before it manifests itself elsewhere.

During systole, the function of the musculature of the arterial wall is to raise the arterial tonus which (1) stimulates the ventricle by increasing the peripheral resistance, and (2) accelerates the velocity of the blood by diminishing the calibre of the arteries ; and during diastole, the aortic end of the arterial system being closed, it is conjoined with the elastic recoil in maintaining the onward flow, the velocity of which is enforced by further contraction. The arterial musculature is therefore an important factor in accelerating velocity throughout the whole cycle of the arterial blood-pressure. When the arterial wall becomes less extensible, either from textural changes or from a supernormal blood-pressure, its muscular fibres develop.

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[1] "Marked tortuosity means that the arterial tone, which resists elongation as well as transverse expansion, has been deficient either absolutely or relatively to the internal pressure. An artery with deficient tone may elongate and become tortuous with normal internal pressure, while another, even with increased tone, may, after a time, yield to excessive internal pressure." (J. A. MacWilliam and J. E. Kesson, *Heart,* vol. iv, 1913, p. 309.)

In support of this conclusion, we possess a fair amount of evidence—post-mortem and clinical—and doubtless further post-mortem observation on the musculature of the arteries in cases of hypertension will substantiate and amplify that evidence. Sir G. Johnson and Howslip Dickinson held this view, and among more recent writers H. D. Rolleston[1] has shown that there is a generalised increase in the arterial muscular fibres in renal disease, in which arterial hypertension is a prominent clinical feature; and T. D. Savill[2] demonstrated the existence of hypertrophy of the muscular tissue of the arteries, designated by him "arterial hypermyotrophy," in cases similar to those described by Sir Clifford Allbutt[3] under the term "hyperpiesis." Clinical observation also supplies us with some data which show that those in whom the arterial pressure is supernormal, whether associated or not with arterio-sclerosis, are more particularly liable either to a generalised temporary increase of arterial contraction, which, for the time, raises the blood-pressure to a still higher level, or to a localised spasm of an artery which induces more or less transient disturbance in the brain, or in the limbs.

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[1] *Lancet*, 1895, vol. ii, p. 137.

[2] *British Med. Journal*, 1891, vol. ii, and 1897, vol. i.

[3] The Lane Lectures, *Philad. Med. Journ.*, 1900, pp. 400–500.

This proclivity to spasm in the arteries is probably an outcome of the rich endowment of muscular fibres in the hypertensive arterial wall. The exacerbations of arterial pressure produced by increments of generalised arterial tonus are too well recognised to require further comment. But I shall discuss a little further some of the varied manifestations of localised arterial angio-spasm, because they are more expressive of the abnormal activity of the hypertrophied muscular tissue of the individual artery than are the effects of general arterial constriction.

The characteristic feature of these disturbances is their transient nature and their liability to recurrence, complete recovery taking place in a few minutes, or in an hour or two, or in a day. They cannot therefore be caused by a lesion, such as hæmorrhage, or a thrombus, or an embolus, which requires some time to elapse before its effects are effaced when improvement does take place. The transitoriness of these disorders, therefore, suggests a functional cause as fugitive as the ailments themselves; and all those who have reported their cases (such as G. Peabody,[1] W. Russell,[3] F. H. Edgworth,[3] Sir W. Osler,[4] O. K. Williamson,[5] M. O. Hunter,[6] Stanley Blaker [6]) are unanimous in attributing them to a spasm in the artery supplying the affected part, which consequently becomes ischæmic. Arterial spasm in the legs may cause intermittent clonus; or senile nocturnal cramp; or a spasm of the middle cerebral artery, or one of its branches may induce slight transient attacks of aphasia, hemiplegia, or monoplegia (affecting only the face, hand, or arm), convulsive attacks with or without loss of consciousness, or with or without clonic spasm in the paralysed part. Sir W. Osler thus classifies these cases:

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[1] A contribution to the "Symptoms and Pathology of Endarteritis Obliterans," Practitioners' Society of New York, 1886.

[2] "Arterial Hypertonus, Sclerosis, and Blood-Pressure," *Encyclop. Med.*, 1907; "Cerebral Manifestations of Hypertonus in Sclerosed Arteries," *Practitioner*, 1906.

In a recent lecture (*Lancet*, 1914, vol. i., p. 1057) W. Russell brings forward an interesting series of cases of cerebral angiospasm producing disturbance of vision in migraine (retinal angiospasm), loss of memory (senile), epileptoid attacks simulating fainting, mental obscuration with attacks of greater obscuration, and inability to work, and temporary inability to perform ordinary work.

[3] "Transitory Hemiplegia in Elderly Persons," *Practitioner*, 1909.

[4] "Transient Attacks of Aphasia and Paralysis in States of High Blood-Pressure and Arterio-Sclerosis," Canadian Med. Association, October 1911.

[5] "On Arterial Spasm" (especially in explanation of certain cases of transient Hemiplegia and Monoplegia), *Practitioner*, July 1913.

[6] Quoted by O. K. Williamson.

"(*a*) Healthy individuals with high blood-pressure, but without signs of arterial disease.

"(*b*) Patients with well-marked arterio-sclerosis, in whom the cerebral attacks have come on without warning, sometimes as the signal symptom. A majority of my cases come in this group.

"(*c*) In advanced sclerosis with cerebral changes, manifested by progressive mental and muscular weakness, all possible types of these transient seizures, including convulsions, may occur. The attacks are most frequent in the aged, but men in the 5th and 6th decades are also affected."

These and other clinical experiences show that hyperplasia of the arterial muscular tissue, though in its physiological degrees conservative and auxiliary, when advanced, may originate more or less grave pathological disturbances.

CHAPTER III

FUNDAMENTAL DATA (*continued*)

III. PULSE-PRESSURE

EACH normal cardiac cycle (systole and diastole) has its counterpart in the arteries wherein the blood-pressure rises to a maximum (systolic) and falls to a minimum (diastolic) point.

Though the duration of the cardiac and arterial cycles is identical, their pressure curves are quite different. The pressure curve of the ventricle, which discharges its load after the manner of a pump, rises from and returns to zero[1]; whereas that in the arteries starts from and gradually declines to a certain residual pressure, which is considerably above the zero point. The grade of pressure in the arteries on which the ventricular pressure is superposed is the "constant" arterial pressure, and is denominated the arterial diastolic, or minimum pressure; and this, *plus* the superadded ventricular, is the arterial systolic, or maximum pressure.

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[1] To be quite correct, the minimum pressure in the ventricle is a minus pressure, *i.e.* it is below zero. (W. D. H.)

The term "pulse-pressure" is employed to express in mm. Hg. the range of the arterial pressure comprised between these limits.

The Normal Pulse-Pressure.—According to J. A. MacWilliam and G. Spencer Melvin,[1] employing the auditory method, the average pulse-pressure in a series of normal adults was 46 mm. This figure practically agrees with my own (45 mm.) for subjects below the middle period of life; but for those over forty or so, my observations show that beyond that age the average range of pulse-pressure increases. It is difficult to formulate a rule for estimating this increase, because the individual differences are much greater after than before the attainment of this period of life. For example, in one subject a 45 mm. range may be retained until the ages of fifty, fifty-five, or sixty are reached; whereas in another a perceptible lengthening of the range may be observed at forty-five or fifty. Therefore, any rule founded on averages is much less trustworthy for individual cases over forty than before that age. It may, however, be stated that the average increment of the normal pulse-pressure is much less between forty and sixty than it is after sixty; and a rough working rule for its application to individual cases may be formulated by adding to the average before forty (*i.e.* 45 mm.) 1 mm. for each two years from forty to sixty, and 1 mm. per year after sixty. On this basis a healthy subject of seventy may have a pulse-pressure of 65 mm. (*i.e.* 45 × 20) without being abnormal.

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[1] *British Med. Journ.*, 1914, vol. i, p. 693.

The Relation of Pulse-Pressure to the Normal Senescent Change in the Circulation.—After the meridian of life (*circa* forty) is attained, a gradual rearrangement of the fundamental factors which determine the arterial pressure takes place sooner or later; and this is the normal outcome of the efforts of the physiological work of the heart, and of the arteries. The alteration doubtless begins in the arterial wall, which becomes less elastic and more muscular (see p. 37). The elastic recoil of the aorta during diastole being reduced, and the peripheral resistance increased, the heart is called upon to do more work, and consequently increases in weight as the years advance over fifty (see p. 14). Thus, the pulse-pressure is extended after the middle period of life, and the gauging of it becomes a useful general clinical guide in showing the rate at which the normal change in the arterial wall and the heart incidental to age is advancing—whether postponed, or fairly normal, or premature. The incidence and rate of progress varies greatly in different individuals. For example, the pulse-pressure of early life (45 mm.) may not be exceeded in certain cases at fifty, fifty-five, or even sixty; and when in such cases the pulse rate is not subnormal, and the heart shows no sign of failure, we may presume that the arteries are preserving their elasticity and suppleness beyond the usual period for average subjects of corresponding age. Then again, the pulse-pressure may even begin to extend before the age of forty; when we may infer that the arterial changes incident to age are setting in prematurely. But before drawing such inferences, we should exercise every care in making our observations to exclude any temporary contraction of the arteries which extends the pulse-pressure.

The Predominant Factors in Determining the Manometric Measurement of the Pulse-Pressure.—The range of the pulse-pressure is the resultant of several circulatory conditions: such as the force, duration, and rate of the ventricular contraction; the volume of the ventricular output; the volume of the blood, the resistance of the blood (viscosity) and of the arteries, arterioles, and capillaries; and the resiliency of the arterial wall. It is hardly possible for the clinician to assign to each of these causal conditions its share in producing the net result; but he can appreciate and measure the leading factors. These are, the force and duration of the ventricular contraction, and the amount of peripheral resistance, which are shown by clinical observation to be closely connected. When, for example, the resistance is reduced the ventricular contractions are less vigorous and of shorter duration and the pulse-pressure is lower than when the resistance is higher. The auditory method of observing the arterial pressure has shown that when the pulse-pressure rises, it is the maximum pressure (the crest of the pulse wave) which takes the lead, and then only after a considerable interval of time does the minimum pressure (the trough of the wave) rise as well. From this fact it would seem that in the first stage of the expansion of the pulse-pressure the ventricle is quite able to surmount the increased peripheral resistance, whereas in the second stage it cannot efficiently cope with it. Hence the clinical importance of reading the minimum as well as the maximum pressure. So long as the expanded pulse-pressure remains in its first stage—the minimum pressure failing to rise—the condition of the arterial circulation may be said to meet the normal requirements for age; but when it passes into its second stage—the minimum pressure being supernormal—the physiological begins to merge into the pathological state.

The Relation of the Pulse-Pressure to the Velocity of the Flow of Blood.—*Cæteris paribus,* a diminished range of pulse-pressure slows the flow of blood through the arteries into the capillaries; whereas an enlarged range increases the velocity of the flow within certain limits (J. A. MacWilliam and G. Spencer Melvin[1]).

The Relation of the Pulse-Pressure to Functional Activity.—It is well known that when the blood is transmitted through perfused organs under rhythmic pressure (pulsation) the effective condition of the tissues is longer maintained, and the rate of perfusion is greater than when the flow is controlled under constant pressure. This fact is but an illustration of what happens in the body, in which rhythmic blood-pressure is a fundamental factor of metabolic and functional activity—the optimum result being attained by a certain range of that pressure. For example, Erlanger,[2] Hooker,[2] and Gesell[3] have shown in regard to renal elimination that the amount of urine and of urea, chlorides, etc., varies directly with the amplitude of the pulse-pressure, and Gesell regards the diuresis of digitalis and strophanthus as the result of the increased range of the pulse-pressure produced by these drugs. We may therefore infer that the measurement of the pulse-pressure furnishes an important tell-tale of the volume of blood supplied to the organs per unit of time.

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[1] *Op. cit.*

[2] *Johns Hopkins Hosp. Reports,* 1904, xii, p. 346 (Erlanger and Hooker); *Arch. of Intern. Medicine,* 1910, v, p. 491 (Hooker).

[3] *American Journ. of Physiol.,* 1913, vol. xxxii, p. 70.

The pulse-pressure is raised and lengthened by:

(1) Organic changes in the arterial wall, as in senile arterial fibrosis and arterio-sclerosis.

(2) Increased tonic contraction of the arteries as in hypertensive ailments and paroxysms (nervous perturbation, migraine, asthma, etc.).

(3) Nephritis (acute and chronic).

(4) Aortic insufficiency.

The pulse-pressure is lowered and shortened by:

(1) Decreased peripheral resistance from reduction of arterial tonus, as in the atrophic or wasting ailments, phthisis, etc.

(2) Anæmia and hæmorrhage from lungs, stomach, intestines, etc.

(3) Cardiac asthenia, collapse, and shock.

IV. PERIPHERAL RESISTANCE

Work consists of movement against resistance. Therefore, the work of the ventricle is performed when it overcomes the resistance offered to the onflow of blood in the circulation. The resistance is minimal in the larger arteries, is gradually increased as the arteries divide, and becomes maximal in the minute arteries and arterioles, in which friction is also maximal. In the ample capillary bed the velocity is diminished, and the resistance is again lessened. The main resistance is in the arterioles and not in the capillaries for the following reason: each individual capillary is small and its resistance therefore great, but their number is so immense and the total bed so large that the resultant resistance offered is comparatively small. Clinicians have not always been in agreement in apportioning the share taken by the various factors concerned in offering resistance to the onflow of blood. For example, in 1876 Sir George Johnson [1] emphasised the clinical importance of localising peripheral resistance in the arterioles, by which it was varied on the "stop-cock" principle; whereas W. Howslip Dickinson,[2] while agreeing with Johnson that the musculature of the arteries and arterioles is increased, held that the resistance is mainly in the capillaries; and Sir William Broadbent [3] regarded capillary obstruction as primary, and hypertrophy of the musculature of the arterioles as secondary. We may, nevertheless, with confidence accept the physiological view that the arterioles form the principal factor in peripheral resistance.

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[1] *Op. cit.*

[2] *Lancet*, 1895.

[3] *The Pulse.*

Is Organic Thickening a Cause of Increased Peripheral Resistance?—During the decadent stage of the life of the arterial wall (*i.e.* after forty) F. W. Andrewes shows that fibrotic proliferation is a prominent pathological feature.[1] It is doubtful, when it pervades the larger and middle-sized arteries only, if it is a factor of importance in increasing peripheral resistance. But should it involve the smaller arteries and arterioles, and reduce the calibre of these vessels, it must raise the arterial resistance in a marked degree. This conclusion is supported by the following experiments of Thoma quoted by F. W. Mott. "Thoma made injection experiments upon cadavers with salt solution, observing the times of injection of a given number of litres at a given pressure when arterio-sclerosis existed, and when it did not. He found it took very much longer to inject the same amount of fluid in the case of sclerosis. Œdema of the lower extremities occurred when only four litres had been injected into a body affected by widespread angio-sclerosis; whereas into the arteries of a body not so affected, seventeen litres could be injected before leakage took place. These and other experiments show that when arterio-sclerosis is present, the salt solution has to overcome much more resistance in the vessels of the lower extremities, although investigation shows that the lumen of the arteries is not greatly diminished. It may be concluded, therefore, that changes in the permeability of the capillary walls are also present in arterio-sclerosis. Does it not also suggest that the capillary area generally is greatly diminished, probably on account of the fibrotic changes referred to?"[1] As in the cadaver vaso-motor action is of course excluded, the results of these experiments indicate that in arterio-sclerosis (1) the peripheral resistance in the smaller ramifications of the arterial system is increased by some structural alteration (probably chiefly fibrotic) and (2) the capillary bed is more permeable than normal and is perhaps diminished in area. It does not follow that vaso-motor action during life is necessarily abolished by the structural change in the arteriolar wall, though in the course of time it may become so, when, as in angio-sclerosis, vaso-dilators can no longer reduce the peripheral resistance and the arterial blood-pressure. This presumed organic reduction of the calibre of the distal arteries suggests why the contraction of the musculature, though of smaller range, is as effective as when the bore was of normal diameter, and why it is unnecessary to suppose that hypertension is maintained for prolonged periods (*e.g.* from year to year) by persistent contraction which is physiologically improbable.

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[1] *Op. cit.*

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[1] *Allbutt's System of Med.*, vol. vi, p. 327.

In 1872 Gull and Sutton [1] demonstrated the existence of a generalised hyaline fibrosis of the peripheral vessels (arterio-capillary fibrosis), of the kidney (primary contracted granular or arteriosclerotic kidney), and other parts (brain, spleen, etc.). J. H. Gaskell [2] shows that the primary change begins in the intima of the renal arterioles and not in the adventitia as supposed by Gull and Sutton. May not a similar pathological thickening be found in the arterioles generally in forms of hypertension not primarily of renal origin?

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[1] *Lancet*, 1872, vol. i, p. 794.

[2] *Journal of Path. and Bact.*, vol. xvi, pp. 287–320. J. H. Gaskell found the internal thickening of the arterioles so great as to almost block the lumen ; and he remarks concerning the view of Gull and Sutton that it "has of late been revived by Jones and Prym (*Virchow's Archiv.*, 1904, Bd. clxxviii, S. 367). They hold that the small artery change is a true arterio-sclerotic process, and is not a special change peculiar to this form of kidney change as some other observers have maintained. They insist also that the small artery change in the kidney is part of a general small artery change affecting many organs. The present investigations bear out this view."

Viscosity of the Blood as a Factor in Peripheral Resistance.—Blood-viscosity is mainly dependent on the percentage of the cellular elements. It is, however, likewise affected by the physical and chemical properties of the plasma, but to a smaller extent. Therefore if viscosity is a prominent factor in increasing peripheral resistance and the arterial blood-pressure, we ought to discover pronounced evidence of it in the various forms of polycythæmia. On this point I quote the following paragraph from F. Parkes Weber's excellent critical review of polycythæmia:[1] "In spite of the excessive viscosity of the blood, the blood-pressure in polycythæmia, though usually, is not always high, and in most cases the left ventricle of the heart is not greatly hypertrophied. Evidently the difficulty in circulation caused by the enormous blood-viscosity is to a great extent avoided by compensatory dilatation of the blood-vessels. This is just what some of the experimental work on the relation of artificially increased blood-viscosity to blood-pressure (Jacobi) would lead one to expect, but all observers are agreed that artificial increase of blood-viscosity does tend to raise the blood-pressure in spite of compensatory dilatation of blood-vessels." Numerical percentages of hæmocytes and heights of blood-pressure are not generally available in the published clinical records of polycythæmic cases; it is therefore difficult to form an estimate of the presumed causal relation between the polycythæmia and the pressure. For example, out of the records of eighteen specially remarkable or obscure cases of polycythæmia comprised in Appendix B of F. Parkes Weber's paper, in only three are found data of blood-pressure; and of these one is ineligible for comparison because of the presence of marked arterio-sclerosis and of albuminuria (though slight). Moreover, in this case the systolic pressure (150–160) is not high in view of age (sixty-four) and of the existence of arterio-sclerosis; so that we can scarcely conceive that over 11,000,000 erythrocytes per c.mm. can have materially, if at all, raised the blood-pressure. Of the two remaining cases, one (aged fifty-one) furnished a systolic pressure of only 100-110 mm. Hg. with hæmocytes over 9,000,000 per c.mm., and the other (an excitable overworked subject aged fifty-five) a pressure of 170 mm. Hg. with hæmocytes of only about

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[1] *Quart. Journ. of Med.*, 1908, pp. 85–134, "Critical Review of Polycythæmia, Erythrocytosis and Erythæmia," by F. Parkes Weber, M.D., F.R.C.P., etc.

6,000,000 per c.mm. In the latter case the proportion of blood-cells was practically only 1,000,000 above normal and the pressure was 60 mm. Hg. higher than in the former case with 4,000,000 in excess.

After excluding the well-recognised factors of hypertension, I cannot say that I have myself been able to discover any correlation between the high readings of my hæmacytometer[1] and of the blood-pressure. And furthermore, the same negative evidence is afforded by J. S. Haldane and his coadjutors on the effects of high altitude (14,093 ft. above sea level) on the blood-pressure and the increased concentration of the blood.[2] Moreover, recent experiments with the heart-lung preparation suggest that increased viscosity of the circulating blood produces cardiac conditions which should lower rather than raise the arterial blood-pressure; for C. Lovatt Evans and Sagoro Ogarva have shown that it reduces the input of the heart which lessens the output and that the smaller ventricular load of concentrated blood is expelled with less endocardial pressure than when it is larger and less viscous.[1]

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[1] See Appendix.

[2] *Phil. Trans. Roy. Soc.*, Series B, vol. 203, p. 185 (1913), "Physiological Observations on Pike's Peak, Colorado, with Special Reference to Adaptation to Low Barometric Pressure," by C. Gordon Douglas, B.M., J. S. Haldane, M.D., F.R.S., Yandall Henderson, Ph.D., and E. C. Schneider, Ph.D.

Increased viscosity has been ascribed to other causes than polycythæmia; such as calcium retention (Sir James Barr), accumulation of carbonic acid (Sir Lauder Brunton), and colloidal uric acid (Alexander Haig). Sir James Barr asserts that "by the free use of decalcifying agents you lessen the viscosity of the blood and rapidly lower the arterial blood-pressure—in this way we get indirect evidence of the effects of calcium in raising the arterial blood-pressure."[2] Sir Lauder Brunton thus refers to the accumulation of carbonic acid: "When the blood becomes very venous, it will flow with difficulty. Oxygenation of the blood lessens the viscosity, and this accelerates the circulation. When blood would not flow from a vein after venesection, I have found the inhalation of oxygen produce a free flow of blood almost immediately."[3] Alexander Haig, in support of his contention that uric acid in its colloidal form is the cause of increased viscosity of the blood in the capillaries, remarks: "When we remember that in the more severe conditions of collæmia, uric acid can be precipitated in the blood drawn from the vessels in bulk equal to one-fourth or even one-third of that of the red cells, we have no difficulty in believing that a viscid colloidal substance (though more or less invisible till a precipitant is used) must, nevertheless, when present in such quantities, greatly affect the viscosity of the blood and hinder the passage of both cells and plasma through the more minute capillaries."[1]

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[1] *Journ. of Physiol.*, vol. xlix (1915), Proc. Physiol. Soc., Jan. 23, 1915.

[2] *Brit. Med. Journ.*, 1910, vol. ii, p. 1336.

[3] *Therapeutics of the Circulation*, 2nd ed., by Sir Lauder Brunton, F.R.S., 1914.

The views of these three observers are quoted merely to give a complete account of the various opinions held, and not necessarily as implying that they should be accepted without further evidence.

Peripheral Resistance in Relation to Age.—Leonard Findlay[2] has observed that a comparison between the systolic pressure in the middle phalanx of the finger (*à la* Gärtner) and arm (*à la* Riva Rocci) in eighty-two normal subjects, varying in age from two and a half to fifty, displays a difference which increases with each decade. After tabulating his individual observations, he remarks: "A most striking feature of these tables is the gradually increasing difference between the so-called central, or brachial pressure, and the peripheral, or digital pressure. During the first decade the average difference amounts to 4·6 mm. Hg. In the majority of the cases the brachial reading is higher than the digital, though in 20 per cent. the opposite is found. Between ten and twenty years the difference rises to 8 mm., and in only 5 per cent. of the cases [one in eighteen cases] is a higher peripheral reading obtained [and that only 5 mm., which is within the limits of error]. From twenty to thirty years the difference is still higher, registering for men 22·1 mm. and for women 13 mm. [or 17·5 for men and women], and for every case the brachial pressure recorded the higher register. In the case of men between thirty and forty years the average difference is 32 mm., and for men between forty and fifty years 44·5 mm. It is thus seen that the older the individual and the higher the blood-pressure, the greater is the difference between the central and peripheral readings." In all cases over twenty the digital differences were *minus*. The following table embodies these results:

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[1] *Medico-Chirurg. Trans.*, 1906, vol. lxxxix, p. 208.

[2] *Quart. Journ. of Med.*, vol. iv, 1911, pp. 489–97, "The Systolic Pressure at Different Points of the Circulation in the Child and the Adult," by Leonard Findlay (from the Physiol. Lab. Glasgow Univ.).

Number of Subjects.	Decades.	Average Differences in mm. Hg.	Percentage of Cases in which Digital Pressure was		
			+	−	0
30	2½–10	4·6	16·6	46·6	36·6
18	10–20	8·0	5·5	66·6	27·7
13	20–30	17·5		100	
11	30–40	32·0		100	
10	40–50	45·0		100	

This observer infers from his experimental production of vaso-constriction by tyramine in man and adrenaline in animals, that the *minus* tactile readings are caused by increased arterial tonus; and this view is emphasised by the still greater peripheral declensions of pressure, such as 100 (nephritis aged thirty-five), 95 (acute Bright's aged forty-four), 70 (nephritis aged thirty-two), which he has observed in hypertension.

From these data it may be inferred that during childhood and youth the systolic brachial and digital pressures are practically uniform; but in adults the digital pressure progressively falls below the brachial as age advances. In other words the pressure gradient from the brachial to the phalangeal arteries is practically negligible in childhood and youth; but develops markedly and progressively in adult life.

The only objections that can be raised against the unreserved acceptance of these results are the defects of Gärtner's method. Hitherto all observations have shown that as an independent method for estimating the arterial pressure it is valueless, because the peripheral site of observation (the middle phalanx of the finger) is anatomically and physiologically unsuited for the purpose. It may, however, fall into its right place when employed for the measurement of the peripheral pressure only; providing, of course, that its well-known defects in technique are met. Then it may serve as an adjunct to the ordinary procedure for determining the brachial systolic pressure; for it will then afford an approximate measure of the peripheral resistance in hypertension, and Leonard Findlay's figures may be taken as a working basis to represent the normal average age differences. As, however, close readings of these differences are not at all necessary, we might even accept a wider rule, such as 30 mm. for normal subjects from twenty to thirty and 60 mm. for those over thirty.

The technique is described later.

Peripheral Resistance in Relation to the Heart.—Peripheral resistance stimulates the ventricle by reflex nervous agency and by fluid conduction which raises the aortic blood-pressure; and is an important factor in eliciting the optimum efficiency of the heart muscle, which, like all muscle, is maintained at its best in energy and bulk by work; hence it is that the heart progressively grows in weight proportionately to the increase of peripheral resistance due to advancing age (p. 14). The rise of the arterial pressure and especially of the diastolic pressure evoked from the competent ventricle by a rising peripheral resistance must make for efficiency of the coronary circulation and the maintenance of cardiac nutrition.

Turning from the physiological to the pathological field we meet with slack arteries, low arterial pressures, and weak ventricles on the one hand; and on the other with tightened-up vessels, high arterial pressures, and hypertrophied ventricles. In the latter class of cases the nutrition and vigour of the ventricle respond for a variable length of time to the supernormal resistance ahead, and the balance thus established is satisfactory from the standpoint of an efficient circulation. But when the resistance can no longer be relieved by treatment, or when it overbears or outlasts the reserve capacity of the ventricle, the effective life of the latter is shortened by the continued high grade of work it is called upon to perform.

At one time cardiac pathologists were dominated by the impression that heart disease originated in the heart itself and that the detection of a murmur was the surest sign of the localised disease. It took many years to correct this error and to widen the outlook so as to embrace the periphery of the circulation in the purview of causation. Now as a matter of course we regard the peripheric and the centric origin of chronic heart disease as of equal importance; and we go further than that, for we recognise the pernicious influence of the stress produced by increased peripheral resistance on disease originating in the heart itself and we endeavour to modify it.

The Measurement of Peripheral Resistance.—The method of determining the brachio-phalangeal pressure gradient affords an approximate measure of the peripheral resistance. Attempts have, however, been made to obtain direct readings from the distal portion of the circulation. The methods which have hitherto been suggested for this purpose are founded on two different principles ; one being to measure the pressure required to blanch the skin under a piece of glass, and the other to determine the time required for the recovery of colour in a spot rendered pale by pressure. The instruments of N. von Kries [1] and Ludwig and of von Basch [2] follow the first principle and are therefore designed to measure the capillary blood-pressure; and the inverted spring balance of Alexander Haig [3] is based on the second principle and is devised to determine the velocity per second of the capillary circulation. Presuming that the onflow from the capillaries into the veins is normally free, the readings obtained by all these pieces of apparatus are mainly if not entirely dependent on the arteriolar blood-pressure and velocity, and are therefore influenced by gravity and by coldness of the skin; hence observation must be made on the heart level and on hands normally warm. The objection to the employment of instruments applied to the hands and fingers with the view of measuring the blood-pressure and velocity of the peripheral circulation is the liability to error from temperature variations. I have therefore avoided direct instrumental observation on the distal vessels, except in the case of that on the first phalanx which yields the arterial pressure gradient. In Alexander Haig's method the skin of the backs of the hand or fingers is paled by the circular pad (10 mm. in diameter) of the instrument under a known pressure; and the capillary reflux is measured by a metronome beating half seconds. Sir James Barr uses a rod of the same diameter and estimates the reflux by a stop watch recording fifths of a second.[1]

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[1] *Ludwig's Arbeiten*, 1875, p. 69.

[2] *Arch. des Scienc. biol. de St. Petersb.*, 1904, vol. xi, suppl. pp. 117-36.

[3] *Op. cit.*

I have found that observation of the effect of gravity on the peripheral circulation in the hand and fingers is quite as instructive as the distal instrumental methods. When an arm is raised to the vertical position with the hand and fingers extended, the blood drains away from the veins and capillaries, so that the former collapse and the hand grows much paler than the other resting on a table. On raising an arm in this way for one minute and then rapidly placing the raised hand by the side of the other (the palms of both resting on the table), we obtain two criteria of the velocity of the capillary circulation, namely, the time required for refilling of the veins, and the restoration of colour in the skin. In normal subjects from one to three seconds elapse before a vein begins to refill or the colour returns. When the veins are not embedded I prefer the venous criterion, as it is more definite than the restoration of colour. In some cases the colour does not fade at all, or but very slightly, though the veins collapse and refill very slowly ; in such cases the venules are probably contracted. When pallor is produced and the veins fill slowly, either the *vis a tergo* is deficient or the arterioles are much contracted ; then the manometer will decide which of these causes of delayed reflux is in operation.

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[1] *Op. cit.*

V. The Storage of Blood in the Veins

The large calibre of the splanchnic [1] and systemic veins, far in excess of that required for the transit of the average volume of blood circulating through them, serves an important physiological purpose—namely, to provide for the temporary storage of blood. During exercise, the systemic veins become loaded with blood, and their reserve capacity enables them temporarily to retain such portions of the blood as the heart may not for the moment be able to discharge into the arteries. In the intervals of rest, the blood employed in systemic work reverts to the capacious splanchnic reservoir, whence it is again withdrawn by the physiological activities of the system. In this fact we find the *raison d'être* of the large capacity of the splanchnic veins, which form an important adjunct to the systemic circulation, serving as a reservoir for the supply of blood needed for the performance of work—whether cerebral or muscular—and as an overflow chamber in times of rest.

In health a certain degree of functional interchange is maintained between the two systems (splanchnic and systemic), so that the " give " and " take " of each fall within moderate limits ; but in disease this normal balance is apt to be greatly disturbed. In one group of cases (the hypotonic) the splanchnic reservoir is continuously surcharged, while the systemic " draw " is lessened, and the arterial pressure is lowered. In another group (the hypertonic) the splanchnic reserve is greatly reduced, and the arterial pressure is raised. The undue draining of blood into the capacious splanchnic venous system and its lodgment there, is a large factor in lowering the arterial blood-pressure in certain diseases, such as phthisis, neurasthenia, toxæmia, convalescence, and other atonic states ; and this result is attributed [1] to the inhibition of tone in the splanchnic arterioles through the path of the depressor nerve (the function of which was discovered fifty years ago by Ludwig and Cyon[2]), and vaso-motor centre. And when the splanchnic arteries and arterioles become the seat of hypertonus or sclerosis, as frequently happens in cases of hypertension, the systemic arterial pressure rises to still higher levels. Contraction of the splanchnic area is, however, not the leading cause of hypertension: it merely increases it just as its relaxation lessens it.

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[1] We are indebted to the demonstration of Leonard Hill for much of our knowledge of the large capacity of, and the part played by, the splanchnic circulation: see "Influence of the Force of Gravity on the Circulation of the Blood," *Journ. Physiol.*, 1895, vol. xviii, p. 15.

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[1] " Experiments on Venous Blood-Pressure and its Relation to Arterial Pressure in Man," by Henry Sewill, M.D., etc., in *Journ. of the American Medical Assoc.*, 1906.

[2] *Arb. aus d. Phys. Anstall.*, Leipzig, 1866, p. 128.

VI. Bio-chemical Causes of Hypertension and Hypotension

The maintenance of hypertension and hypotension mainly rests with the musculature of the arteries, their contraction and relaxation, rather than with such mechanical causes as reduction of the calibre of the arteries by fibrosis or atrophy of the arterial wall. We cannot refer continuous alterations of the arterial blood-pressure to changes in the non-muscular textures of the arterial wall. We therefore look for some bio-chemical agent or agents which actuate the arterial muscle fibres.

Chemical Causes of Hypertension.—There is no evidence to show that any of the most common metabolic products, such as urea, uric acid, xanthine, etc., can induce a state of hypertension. But we know of three pressor substances generated within the body, any one of which is of sufficient activity to maintain the condition of hypertension of the cardio-arterial muscle fibres, provided it is continuously supplied to the blood in the required quantity. They are derived respectively (1) from the medulla of the adrenals, (2) from the infundibular portion of the pituitary body, and (3) from putrefaction of proteins in the intestines.

(1) In France there is a prevalent belief among clinicians that superadrenalism is the *vera causa* of hypertension: a belief largely founded on the work of Josué,[1] which shows that the injection of adrenaline into rabbits produces aortic atheroma and a rise of arterial pressure.

Experimental research demonstrates that, so far as the cardio-vascular system is concerned, the physiological rôle played by adrenaline is two-fold: namely, (1) to maintain in conjunction with the sympathetic nerves the normal tone of the heart and arteries, and (2) to serve as a chemical messenger to further brace up the circulatory musculature in conditions of stress, such as fright, worry, sudden efforts (fighting, etc.). For the maintenance of tone the discharge of adrenaline is slow and more or less continuous though fluctuating according to requirement, and to the rate at which the store of it in the suprarenal medulla is replenished; but during psychological and physical emergencies it has been shown (especially by the work of T. R. Elliott) that the unloading of it is rapid, and the chemical messenger does its work on the cardio-vascular system almost simultaneously with the execution of the direct nervous impulse on the heart and arteries, which therefore receive the impact of two synergic stimuli. We infer that adrenaline under the ordinary conditions of life is an important factor in maintaining the tone of the musculature of the heart and arteries [1]; this is therefore an inference, but it is a legitimate one. But the unloading of it by stimulating the sympathetic and the presence of the discharged adrenaline in the blood have been clearly demonstrated.[2]

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[1] *Traité de l'Artério-Sclerose,* par O. Josué.

Whether there be a condition of superadrenalism, which expresses itself clinically as hypertension and cardiac hypertrophy, is another matter. So far the proof of this causal relation is not forthcoming. Concordant changes in the adrenals of subjects suffering from hypertension have not been demonstrated; and the experimental evidence is equally negative.[1]

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[1] Adrenaline is a remarkable sensitiser of muscle. For example, W. Burridge finds that a heart perfused by solutions of inorganic salts becomes less sensitive to the calcium present in them, but is sensitised by the addition of adrenaline (1 in 10,000,000), which may even quadruple the duration of its activity (*Journ. of Physiol.*, vol. xlix, 1914, Proc. Phys. Soc., p. lxi).

[2] The reader should study T. R. Elliott's instructive work in his article on "The Control of the Suprarenal Glands by the Splanchnic Nerves," *Journ. of Physiol.*, vol. xliv, pp. 374–409, and his Sidney Ringer Memorial Lecture, *Brit. Med. Journ.*, 1914, vol. i.

The recent work of Douglas Cow,[2] which has demonstrated the existence of a direct vascular connection between the suprarenal medulla and the kidney, may ultimately throw some light on this question. Hitherto we have formed our conception of the physiological rôle played by adrenaline from the passage of the whole of this product into the general circulation; now we find that an appreciable portion of it is also discharged directly into the kidneys, in which it regulates urinary activity—under certain conditions diminishing the excretion and flow of urine. It was found that clamping the adrenal vein completely arrested the flow of urine—the adrenaline being diverted in excess to the kidney. The proportion of adrenaline normally supplied directly to the kidney is doubtless small compared with that discharged into the general circulation; but it will be relatively large, because it is not subjected to the dilution and oxidation of the systemic discharge. The outcome of this inquiry of Douglas Cow opens up the question as to how far the pathological rôle of adrenaline may be ascribed to its suppressive control of renal work and the gradual reduction of kidney substance.

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[1] *Journ. of Exper. Med.*, vol. xvi, 1912, pp. 541–57, "The Question of Epinephrine in the Circulation and its Relation to Blood-Pressure," by Theodore C. Janeway, M.D., and Edwards A. Park, M.D. These experimenters conclude "that there is no evidence that epinephrine [a synonym of adrenaline] in amounts sufficient to produce its physiological effects upon any hitherto test objects, exists in the circulating blood, with the exception of blood from the suprarenal vein. The examination of uncoagulated blood from six persons with high blood-pressure has failed to show the presence of epinephrine or other constricting substance."

[2] *Journ. of Physiol.*, vol. xlix, 1914, pp. 443–52.

Apart from the question of causation, may not adrenaline disturb the course of hypertension? For many years I have been impressed with the fact that cases of hypertension, and especially cases drifting towards it, are often much more prone than others to a supersensitive state of the vasomotor mechanism and temporary increments of arterial pressure from nervous and other causes of disturbance; as if, in such cases, the direct stimulus of perturbation on the circulation is enforced by the undue unloading of adrenaline. In many cases of hypertension high manometric readings when communicated to the patient are apt to produce a state of great nervous apprehension, which aggravates the hypertension. In such cases the manometer in the hands of an incautious and injudicious practitioner may become an instrument of harassment and misery in which superadrenalism may play an important rôle.

It is also not improbable that the adrenals may influence hypertension in the opposite direction; for when an infective fever sets in, hypertension diminishes very considerably, and the adrenaline load also falls. Many workers, especially of the French school, hold that infections depress, or may even abolish, the secretory work of the adrenals;[1] and in this way they may kill.

(2) The pressor product (pituitrin) of the infundibulum of the pituitary body, though possessing a similar constrictive or tonic action to that of adrenaline on the musculature of the circulatory organs, has not been invoked as a cause of hypertension and there is no evidence to show that it should be so regarded. Its primary physiological objective seems to be to tone the nervous centres and through them to maintain the normal tonicity of the cardiovascular musculature. We have no positive proof of its passage into the blood as in the case of adrenaline; although it could diffuse thence *via* the cerebro-spinal fluid. Unlike adrenaline, it does not accelerate the heart-rhythm, and may indeed slow it; and it dilates the renal vessels instead of contracting them, and thus increases the output of urine.

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[1] See also Mott and Halliburton, *Arch. of Neurol.*, 1907, vol. iii, p. 123.

(3) The recent discovery of some powerful pressor bases in the urine, one of which has been isolated as an oxalate and identified by W. Bain as iso-amylamine, when retained in the blood may, on further investigation, go far to explain how some forms of hypertension with cardiac hypertrophy are produced. This experimenter says,[1] in summarising his conclusions, "The normal urine of adults contains certain bases which, when injected into the blood-stream of anæsthetised or pithed animals, produce a marked rise of arterial pressure; these bases are either reduced in amount or entirely absent from the urine of patients with high blood-pressure. The non-excretion of these bases leads to their retention in the blood, and is an important factor in the causation of hypertension. The bases doubtless arise in the intestines, as a result of the putrefaction of proteins, and their amount can be considerably diminished by a marked decrease in protein intake."

We still await the final proof of this view—the evidence of the presence of these putrefactive pressor bases in the blood.

Cholesterinæmia.—Some Russian and French (Chaffard and his school) pathologists have discovered that the feeding of rabbits with cholesterin

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[1] *Quart. Journ. Expt. Physiol.*, vol. viii, 1914.

(cholesterol) produces in three or four weeks a true atheroma of the intima of the aorta and an enlargement of the adrenal cortex due to an enormous increase of its store of lipoid (fat-like bodies.) T. R. Elliott[1] confirms one of these results—the lipoid engorgement of the cortex. The percentage of cholesterin in human blood has been determined by the Paris workers, who find it low in acute fevers, and especially in pneumonia, in which T. R. Elliott observed exhaustion of the cortical lipoid, and high in arterio-sclerosis and atheroma, in which cholesterin exists in proportionately large amount in the cortex.[2] In dogs the results were less obvious. The French investigators ascribe atheroma, arterio-sclerosis, and hypertension to the presence of an excess of cholesterin in the blood (hypercholesterinæmia). They regard cholesterin in normal percentage as antitoxic and beneficial; but when present in excess they hold that it is precipitated into the tissues of the arterial wall, where it and its lipoid compounds excite contraction of the tunica media (hypertension) and the production of arterio-sclerosis. They have found an excess of cholesterin not only in the blood, but in the arterial wall of arterio-sclerotic and atheromatous patients.

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[1] The Sydney Ringer Memorial Lecture, by T. R. Elliott, M.D., F.R.S., *Brit. Med. Journ.*, 1914, vol. i. Elliott and Tuckett (*Journ. Physiol.*, vol. xxxiv, 332, 1906) appear to have been the first to show that the aniso-tropic globules in the cells of the adrenal cortex are not composed of true fat; Rosenheim and Tebb (*Proc. Physiol. Soc.*, Feb. 27, 1909, *Journ Physiol.*, vol. xxxviii) found that these globules consist of a mixture of free stearic and other fatty acids with cholesterin esters, as well as a phosphatide (sphingomyelin), one of the components of so-called protagon. These observations suggest that the adrenal cortex is concerned in lipoid metabolism. (W. D. H.).

[2] *La Cycle de la Cholesterinémie,* par M. le doct. Grigaut (Paris, 1913).

These conclusions have, however, been recently controverted by the work of C. Cantieri,[1] who has shown that in nineteen cases of arterio-sclerosis, with or without high blood-pressure, cholesterin was present in the blood in rather less than the normal percentage (0·175), that there was no connection, even in acute and chronic nephritis, between the height of the arterial blood-pressure and the quantity of cholesterin present in the blood (thus confirming the observations of some other investigators), and that the administration of cholesterin as a drug does not raise the arterial blood-pressure.[2]

Nephritic Hypertension is generally more pronounced than other forms. I shall quote the conclusions of two recent studies of hypertension in nephritis: one by Theodore C. Janeway,[1] and the other by H. Batty Shaw.[2] Janeway writes:

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[1] *Riv. Crit. di Clin. Med.*, Florence, 1913, vol. xiv, p. 657.

[2] Dixon and Halliburton (*Journ. Physiol.*, vol. xlvii, 229, 1913) have also shown that the rise of pressure produced by injecting cholesterin is negligible.

"Hypertension may arise through purely quantitative reduction of kidney substance below the factor of safety. It is difficult to conceive of this as other than a vascular hypertonus due to retained poisons of some kind. Its clinical paradigm is the hypertension accompanying bilateral ureter obstruction, or the unfortunate surgical removal of the only functionating kidney. Possibly it is one factor which helps to produce hypertension in the contracted kidney.

"2. Hypertension may arise in connection with the unknown intoxication which causes disturbances of the central nervous system, and which we call uræmia. This intoxication is not one of retention, in a strict sense, though it is most commonly present in those cases of advanced nephritis which manifest marked nitrogen retention. Clinically, it is related with severe acute nephritis, sometimes at its very onset, and also with subacute and chronic inflammatory affections of the kidney.

"3. Hypertension may arise in primary irritability of the vaso-constricting mechanism from unknown, probably extra-renal causes, which lead eventually to arteriolar sclerosis. In this type the disease in the kidney is the consequence, not the cause, of the generalised vascular lesion. When it progresses to a condition of extreme atrophy, resulting in the true primary contracted kidney, a renal element may be added to the existing hypertension. In some cases arterio-sclerosis of the larger vessels may spread peripherally, and produce a similar form of disease. In these primary vascular diseases it is probable that eventual widespread narrowing of the arterial stream-bed in some cases produces a permanent organic increase in peripheral resistance.

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[1] *American Journ. of the Med. Sciences*, 1913, vol. cxiv, p. 625: "Nephritic Hypertension: clinical and experimental studies," by Theodore C. Janeway, M.D., M.A.

[2] *Brit. Med. Journ.*, 1910, vol. ii, p. 1761.

" What are the vascular poisons back of these types of hypertensive disease ? That question no one can answer. That epinephrine (adrenaline) is one of them, is possible; that it is the only one, seems to me improbable. One may say the same for the secretion of the hypophysis (pituitary). I believe it is likely that different poisons produce different types of hypertension. One toxic case we can name with certainty, lead. Excessive stimulation of the central vaso-motor mechanism must also play some part in producing the varied clinical picture.

"The first and second types of hypertension may at any time be superimposed upon the third. While the second, the uræmic type, may be considered dangerous in itself, hypertension in the arterio-sclerotic or athero-sclerotic kidney is best regarded as a compensatory effort of the organism, as Bier first suggested, to be interfered with only when danger threatens, either of cardiac failure or of cerebral hæmorrhage."

I now quote H. Batty Shaw: "During the past four years, owing to the generosity of my colleagues in the hospital (University College), I have had access to all the cases which have revealed hypertension. Of this number, which was very large, twenty-nine cases have proved fatal. In every case the heart was shown *post mortem* to be hypertrophied; in every case there was general arterio-sclerosis; *in every case there was fibrosis of the kidneys*, some were smaller than normal, some were of normal size, some were larger than normal. The sequence of events is (1) loss of kidney substance, (2) hypertension, and (3) arterio-sclerosis and cardiac hypertrophy."

Chemical Causes of Hypotension.—It is possible that in some forms of hypotension fatigue products are generated in the body; and perhaps these may be isolated and identified in course of time. But depletion of the pressor products (by infective fevers, or by fright, or by a condition of chronic fear, as in neurasthenia) or inadequacy of the secreting cells of the infundibulum or adrenal medulla are more probable causes of hypotension. T. R. Elliott points out that "in many diseases the adrenaline load of the glands is diminished, but not often to what might be looked upon as a really serious state of depletion. The weight of a single normal gland in man is 4 grammes and its adrenaline load is 4 mg. This falls to 1 mg. in many fatal fevers. The greatest exhaustion that I have seen to occur rapidly was not in fevers, but in cases in which the patient was, so to speak, fighting for life, struggling to keep up his heart. In these the adrenaline load was less than 0·05 mg."

It has been repeatedly observed that an intercurrent febrile condition (infective) such as pericarditis, pleurisy, pneumonia, etc., will lower the arterial blood-pressure in even severe cases of hypertension. In illustration of this fact I am indebted to H. Batty Shaw for the following comment: "It is necessary to warn observers that, even by use of the sphygmometer, they will not be able to find hypertension in every case of kidney sclerosis; one observer might be called in to see the patient when the tension is high, and make a correct diagnosis; another called at a later period, when infection has occurred, will find it low, and so miss the true diagnosis."[1]

VII. Physiological Variations of the Arterial Blood-Pressure

In the ordinary routine of life the systolic blood-pressure variations rarely exceed 20 mm. Hg. above or below the normal pressure of the individual; and these limits will be lessened by avoiding the measurement of the pressure for an hour or so after the ingestion of a full meal, or after active or prolonged exercise or effort, or during a highly heated or chilled condition of the body. The influence of meteorological and climatic variations of temperature on the arterial blood-pressure is referred to elsewhere.

A Test of the Integrity of Cardio-Vascular Adjustment for Blood-Pressure.—During exercise, the pressure rises very considerably; but when the heart and the vaso-motor mechanism are quite normal, if the exercise is slight, the pressure returns to normal almost immediately after its cessation. Turning this fact to practical account, I have observed that the integrity of the circulatory mechanism may be tested just after the arterial blood-pressure has been measured. The patient is told to throw the muscles of both legs into a state of rigidity, and at the same time to breathe normally; and while this state of tension is maintained for some two or three minutes, the pressure is again determined. I have observed that in normal subjects the pressures, systolic and diastolic, rise 10, 15, or even 20 mm. Hg., but immediately on the cessation of the muscular tension they fall to their previous levels. I have had but few opportunities for applying this procedure to cases of hypertension and hypotension; but I venture to suggest it to others who have. I have, however, observed rises of 40 or 50 mm. Hg. in hypertension, which require time for their subsidence. From the facts observed, we can realise that the blood-pressure in hypertensive cases must rise to risky levels (sometimes fatal) when the patient strains with closed glottis in defæcation or in other efforts.

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[1] The hypotensive effect of feverishness may also explain why hypotension is sometimes lowered by an intercurrent attack of acute gout.

The hypotensive effect of recumbent rest is well known. It is often well illustrated in hypertensive cases in which the pressure, when the patient is not confined to bed, is lowered by absolute recumbency. In these bed cases, the pressure remains at a lower level as a rule; but in some of them it will rise again. H. Batty Shaw has published the night and morning systolic pressures of at least two such cases.[1] The explanation of this after-rise is not obvious; but I think it may rest with the gradual liberation of a larger amount of blood than usual, lodged in the splanchnic area in the erect postures, forming in recumbency an accession to the volume of blood in the systemic circulation (see p. 66).

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[1] *Brit. Med. Journ.*, 1910, vol. ii, p. 1762.

CHAPTER IV

READING THE ARTERIAL PRESSURE

The trustworthiness of the readings obtained from a manometer depends on the criteria adopted; for these vary considerably in their accuracy, scope, and practicability. The best criterion is that which reduces as much as possible the personal equation of error in the observer, minimises all sources of fallacy, is easily acquired and applied, and is, moreover, sharply defined.

The methods of reading the arterial pressure cycle are the tactile, the oscillatory, and the auditory.

I. The Tactile Method

When the armlet is adjusted to the upper arm, or to the forearm, the finger placed over the radial artery affords a definite reading of the systolic pressure, which is indicated by the first detectable pulsation felt on gradual decompression, after overstepping the obliteration of the pulse by the compression of the armlet. The principal advantages of this criterion are simplicity, general practicality, and its equal availability for observation with the armlet, whether adjusted to the forearm or to the upper arm. But these advantages are somewhat lessened by individual differences in the keenness of the tactile sense: any such defect can, however, be met by training the finger to detect the faintest pulsation, as, for example, when the palpatory method is used in conjunction with the somewhat more delicate auditory method. The deficiency of the tactile method is shown by its inability to furnish a sufficiently precise reading of the diastolic pressure. Many observers must have recognised that, on releasing the armlet pressure, the pulse at the wrist remains small for some time, and then somewhat quickly expands in volume and as quickly subsides again; and I have observed a general agreement between the expansion of the pulse and the point of maximum oscillation of an indicator, which has hitherto been regarded as the visual criterion of the diastolic pressure. F. A. Faught suggests that we should utilise this fact as the palpatory indication of the diastolic pressure. He says: "It will be noted that at first the pulse is very feeble and thready in character, and continues so for a time, when, as the pressure falls, it will suddenly assume the full bounding character of the pulse in aortic regurgitation. At the moment when the change occurs, the height of the mercury column will represent the diastolic pressure." [1]

The method of reading the diastolic pressure should be more precise and definite than that of the systolic pressure, because its smaller variations are in clinical significance on an equality with the more ample ones of the latter pressure. The proposed tactile diastolic criterion, however, falls far short of this requirement, being less definite than that of the systolic pressure.

J. A. MacWilliam and G. Spencer Melvin, relying on their experimental and clinical observations, remark that " methods of estimating diastolic pressure by examination (palpatory or graphic) of changes occurring in the artery peripheral to the armlet (Strassburger, Masing, Sahli, Bingel) are not recommended, the indications being dubious, and their interpretation unreliable." [2]

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[1] *Blood-Pressure from the Clinical Standpoint,* by F. A. Faught, M.D., Philad. and Lond., 1913, p. 51.

[2] *Heart,* vol. v, 1914, p. 194.

II. The Oscillatory Method

(Graphic and Visual)

Oscillation as a criterion of arterial blood-pressure has occupied a prominent position in the evolution of sphygmomanometric methods. In ordinary manometers it has been relied on mainly as the visual indication of the diastolic pressure only; the reading of the systolic pressure being determined by the tactile method. But in graphic recording sphygmometers, such as Erlanger's,[1] Hirschfelder's,[2] Recklinghausen's,[3] Gibson's[4] with Singer's modification, and in the visual sphygmo-oscillometer of Pachon,[5] the behaviour of oscillation under decompression of the armlet is accepted as the criterion of both pressures.

The Oscillatory Systolic Criterion.—As oscillation cannot be totally abolished as a rule by compression, even though this is pushed to 50 or more mm. beyond the obliteration point, the observer must rely on another indication of the systolic pressure than the abolition of pulsation. He reduces the pulsation to a uniform minimum size, and on decompression he notes the point at which it suddenly increases. This is the systolic-oscillatory reading *à la* Erlanger [1] (following Recklinghausen [2] and Pachon [3]). With regard to this index, G. Spencer Melvin and J. R. Murray observe: "Not infrequently the oscillations are developed in such a way as to make it impossible to fix upon any phase as the systolic index. Many observers have found difficulty in regard to this index. In a recent investigation (on healthy subjects) Weysse and Lutz [4] were unable to make a reading in 37 per cent. of their tracings." [5] According to this criterion Pachon's instrument yields much higher readings of the systolic pressure than those afforded by the tactile, or the auscultatory method: this discrepancy is not, however, due to greater delicacy in recording the pressure, but is attributable to (1) the armlet being below the standard width (12 cm.), (2) the ill-defined nature of the criterion itself, and (3) the transmission of vibrations derived from the beats of the distended artery on the upper border of the armlet.

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[1] *American Journ. of Physiol.*, 1902, vol. vi, p. 22; 1904, vol. xi, p. 14; *Johns Hopkins Hosp. Rep.*, 1904, vol. xii, p. 53.

[2] *Diseases of the Heart and Aorta*, 1910, p. 21.

[3] *Archiv f. exper. Pathol. u. Pharmakol.*, 1906, vol. lv, pp. 375 412

[4] *Proc. Roy. Soc. Edin.*, 1907–8, vol. xxviii, pp. 343 *et seq.*

[5] *Comp. Rend. de la Soc. de Biol.*, 1909, vol. lxvi, pp. 723-6.

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[1] *Johns Hopkins Hosp. Rep.*, 1904, vol. xii, p. 53; *Amer. Journ. Physiol.*, vol. xxi; *Proc. Amer. Phys. Soc.*, p. xxiv.

[2] *Arch. f. exp. Path. u. Pharm.*, vol. xlvi, p. 78; lv, pp. 375–412.

[3] *Brit. Med. Journ*, 1910, vol. ii, p. 1765 (art. by K. E. Eckenstein, M.B., Lond., etc., on "The Estimation of Blood-Pressure by means of the Sphygmo-oscillometer of Pachon").

[4] *Amer. Journ. Physiol.*, 1913, vol. xxxii, p. 427.

[5] *Quart. Journ. of Exp. Physiol.*, 1915, vol. viii, pp. 125–39.

The Oscillatory Diastolic Pressure.—The interpretation of this pressure by the oscillatory method is also a matter of uncertainty. For example, Erlanger takes either the mid-maximum, or the lowest maximum point, and Recklinghausen selects the latter, whereas Gibson prefers the former, and Pachon accepts the sudden diminution in the size of the maximum oscillations as the criterion of the diastolic pressure.

According to J. R. Murray's observations, the diastolic criterion of Pachon corresponds closely with the auditory results "when the sphygmo-oscillometer gave quite unequivocal indications (which was not always the case)."[1] As the auditory criterion of the diastolic pressure has been verified (see p. 97), we may, from these observations, conclude that Pachon's indication is a nearer approximation to the actual diastolic pressure than the Erlanger-Recklinghausen criterion, founded on the maximum oscillation. I therefore suggest its use in those exceptional cases in which the auditory method is not applicable, and its adoption by observers debarred from the auditory method by defective hearing. It can be quite easily employed on a mercurial manometer possessing a 3 mm. bore, as in my instrument (see p. 5).

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[1] *Brit. Med. Journ.*, 1914, vol. i, p. 699.

But in attempting to measure the arterial blood-pressure by oscillatory criteria, we are apt to exclude from consideration the disturbing influence of the arterial wall, which observation shows to be very considerable. In practice we meet with two well-defined groups of cases which illustrate the behaviour of oscillation under varying degrees of compression. In one group we can define with comparative ease the maximum region of oscillation, its middle and lower point, or the drop below it: and in cases of this kind we generally find the pressure normal, or perhaps somewhat subnormal, with soft compressible arteries in subjects of under thirty or thirty-five. In the other group, the oscillations on decompression increase suddenly high up on the scale, and remain large over a considerable range in which it is difficult to define the exact point at which they diminish; and in cases of this type the systolic pressure may or may not show an increment for age, and the brachial artery is often thickened and sometimes unevenly so. If, in such cases, we rely solely on oscillatory criteria, the interpretation of them is often one of considerable uncertainty. The recent experimental work of J. A. MacWilliam,[1] J. E. Kesson, and G. Spencer Melvin throws considerable light on the probable causes which produce the clinical anomalies of the oscillation method; these are (*a*) the distensibility and flexibility of the arterial walls; (*b*) the time available between the beats; (*c*) the form of the internal pressure curve; and (*d*) the part played by elongation of the vessel at each beat. In reference to the effect produced on oscillation by differences of resistance along the course of the artery under compression, these observers remark: "The occurrence of maximum oscillation at or near the systolic pressure (pulse obliteration) seems paradoxical. But such is quite conceivable in the case of any artery which is very compressible near its distal end, while resistant and non-distensible (*e.g.* from strong contraction) along the rest of its length. With such an artery in the compression tube, we have sometimes seen the maximum oscillation approaching the systolic (obliteration) level in a remarkable way: the compressible distal end was closed by an external pressure not much above what was necessary to elicit maximal pulsation in the rest of the tube." [1]

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[1] *Heart,* vol. v, 1914, p. 153: "The Estimation of Diastolic Blood-Pressure in Man," by J. A. MacWilliam, M.D., Regius Prof. of Physiology, Univ. of Aberdeen, and G. Spencer Melvin, M.D., Asst. in Physiology, Univ. of Aberdeen; *Seventeenth Internat. Congress of Med.*, London, 1913: "The Estimation of Systolic and Diastolic Blood-Pressure," by Prof. J. A. MacWilliam, J. E. Kesson, and G. Spencer Melvin.

A glance at a sphygmomanogram (the record of oscillation) suffices to show us how indefinite are the readings of the systolic and diastolic pressures. Moreover, it does not possess the redeeming features of an ordinary sphygmogram, which often provides useful clinical information.

III. THE AUSCULTATORY METHOD

In 1905 Korotkow [2] of Petrograd, guided by experimental data, proposed the use of the stethoscope for determining arterial blood-pressure in man; and Krylow,[3] a physician in Petrograd, was the first to adopt it in clinical observation. The method was favourably accepted, and freely employed in Russia and Germany; and after the lapse of some years it was taken up by a few observers in the United States. But in 1910, not having heard of its adoption by any British observer, I ventured to bring forward the results of my own observations, and to suggest certain modifications in technique.[1]

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[1] *Heart*, vol. v, 1914, p. 172.

[2] *Mittheilungen der Kaiserl. Milit-Mediz. Akad. z. St. Petersburg*, 1905, vol. ii, p. 365.

[3] *Verhandlungen des Kongresses für innere Medizin*, Wiesbaden, 1907, vol. xxiv, pp. 404 *et seq.*

More recently the method has been subjected to a thoroughly critical examination, experimental and clinical, by J. A. MacWilliam,[2] Regius Professor of Physiology in the University of Aberdeen, and his assistants J. E. Kesson,[2] G. Spencer Melvin,[2] and J. R. Murray,[3] and the outcome of this investigation not only confirms the main results of previous work, but has established the method on an assured basis.

The Throb.—When the end-piece of a stethoscope is placed over the brachial artery at the bend of the elbow, under pressure a bruit is heard, the quality of which varies with the amount of pressure and the position of the end-piece in relation to the arterial wall, the bruit assuming the character of a throb, or a harsh murmur, or of both. The harsh scratchy murmur can be produced at will by pressing the rim of the stethoscope unevenly over the artery. The bruit is produced by the pressure brought to bear on the end-piece ; a certain degree of pressure is necessary to produce it, and a further degree intensifies it. The armlet is a device which enables us to distribute external pressure evenly over a 12 cm. length of artery, so that, when the end-piece of a stethoscope is applied over the vessel just beyond it, the bruit may be heard free from the factitious disturbance produced by the irregular application of pressure.

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[1] *Proc. Roy. Soc. of Medicine*, 1910, vol. iii (med. sec.), p. 207, and (clin. sec.) p. 8; *Quart. Journ. of Exp. Physiol.*, vol. iv, 1911, p. 45.

[2] *Heart*, vol. v, 1914; *Brit. Med. Journ.*, 1914, vol. i, p. 693.

[3] *Brit. Med. Journ.*, 1914, vol. i, p. 697.

The Cause of Throb.—The production of throb in the artery depends on three leading factors: (1) external pressure, (2) a sufficient velocity of current, and (3) vibration of the arterial wall.

The Site of the Production of the Throb.—It had hitherto been held that the throb is generated in the distal artery just beyond the armlet.[1] J. A. MacWilliam and his assistants, however, have shown that this view is untenable, and that the sounds are produced by vibration of the walls of the artery compressed by the armlet; not much, if at all, by changes in the distal portion of the vessel. I endorse this conclusion, and I regard the pronounced character of the throb over the artery at the bend of the elbow as due to the augmented conduction by current, and not as indicating the place of its origin.

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[1] In 1910 (see *Quart. Journ. of Exp. Physiol.*, vol. iv, p. 46) I upheld the view then current; but since that date I have been led by observation to discard it.

The Phases of Throb.—When the armlet-pressure is raised or lowered, a regular succession of variations of the quality of throb is discovered. On compression, these phases are not so apparent or so loud as on decompression. The release throb is generally sharp and clear, resembling the sounds of a fœtal heart; then this quality of clear and sharp definition gives place to a muffled quality or murmurishness, or a distinct murmur, which is succeeded by a loud and clear note or thud without murmur; and finally, the throb suddenly becomes quite dull and distant, before it vanishes.[1] Putting aside the reappearance and the final disappearance of the throb, decompression furnishes four successive stages of variation in the quality of the sound, in the following order, characterised by (1) sharp-

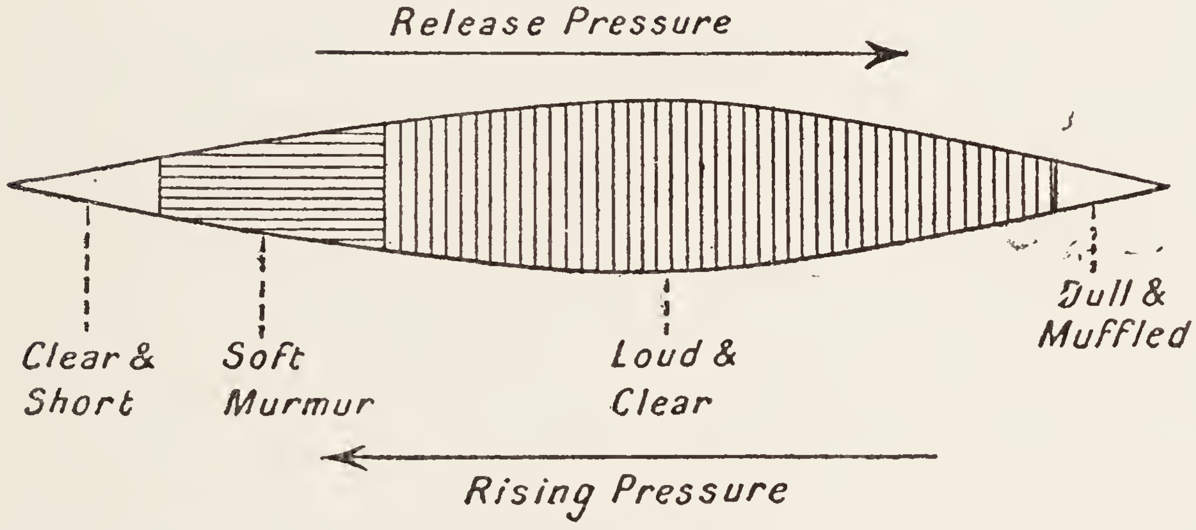

FIG. 5.

By kind permission of the Editor of "The Practitioner."

[1] Ettinger, *Wiener Klin. Wochenschr.*, 1907, vol. xx, p. 992.

ness, (2) murmurishness, (3) loudness, and (4) dulness (see Fig. 5). Of these phases, the murmurish one is the least constant, being frequently absent or so short as to be easily missed. Several observers have assigned some clinical significance to the length of each phase, which varies considerably in different cases. But I think that such an influence is of doubtful value, except in the case of the third phase, the middle third of the range of throb, which I believe to be a useful guide in estimating the tone and vigour of the heart; for when there is cardiac weakness, the range of maximum throb is shortened, and the loudness of it is reduced; and when the vigour of the heart is restored, the range is lengthened and the throb becomes louder. The effect of tonic treatment of the heart can thus be followed.[1]

The Auditory Criteria of the Systolic and Diastolic Pressure.—There has never been any doubt as to the auditory criterion of the systolic pressure. It is the first throb heard on decompression after obliteration. The reading generally somewhat exceeds that of the tactile method—the difference being from 3 to 5 mm. But the diastolic criterion has hitherto always been regarded as uncertain. Most observers have, however, accepted the disappearance of throb on decompression as the best indication of it for all practical purposes, possessing as it does the quality of sharp definition which all observers can recognise without training.

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[1] Fischer, *Zeitschr. f. diatet. u. physik. Therap.*, Oct. 1908; *Practitioner*, 1914, p. 90.

But many observers, including myself, have thought the true diastolic point must be somewhat higher, and have suggested that it should be placed just where the clear, loud note becomes suddenly dull, and muffled or distant. Personally I have found this index precise enough for accurate work, and I have no doubt others will find it to be equally definite.[1] But now, any hesitancy in accepting this as the true auditory diastolic point is removed by the recent investigation of J. A. MacWilliam, G. Spencer Melvin, and J. R. Murray, the results of which I subjoin:[2]

"At a comparatively early stage in our investigation of methods of blood-pressure estimation in man we had recourse to animal experiment, though we have not hitherto published any statement of such experiments having reference to the verification of diastolic pressure estimation by the auditory method."

"In the case of small animals, the testing of the auditory method is not satisfactory, but in animals of sufficient size the procedure offers no special difficulty apart from those connected with respiratory disturbance associated with the anæsthetic. We have found the sheep a very suitable subject. The results are definite and convincing."

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[1] *Practitioner*, 1914, p. 90.

[2] *Journ of Physiol.*, 1914, vol. xlviii (Proc. Physiol. Soc., March 14, 1914), p. xxvii.

"The animal is anæsthetised with ether and chloroform, after a preliminary injection of morphine and chloral. A large-sized reservoir cannula is placed in the central end of the left carotid artery at the root of the neck, and is connected by non-distensible tubing with a set of manometers—valved ones for maximum and minimum pressures and a 'compensated' one for mean pressure—so that the intra-arterial pressure can be brought to bear on any of the manometers as desired."

"An ordinary armlet 12 cm. broad is applied round the neck, it being noted that the circumference of the latter is not too great for such a breadth of armlet. The armlet pressure being raised and lowered in the usual way, the auditory phenomena are examined by means of an Oliver tambour (phonendoscope) applied over the right carotid artery on the distal side of the armlet, the artery being left covered by the tissues with the exception of the skin.

"In order to prevent disturbance from venous congestion of the head due to obstruction caused by the armlet pressure, the internal jugular vein is protected by a split lead tube (15 cm. long) which prevents its being compressed by the armlet pressure. A tracheal cannula was sometimes used."

"One observer made the auditory observations and read off the armlet pressures from a mercury manometer, while another determined the intra-arterial pressures as shown by the valved manometers. The observers frequently changed places, and repeated the estimations. The operative part of the experiments was performed by two of us (J. A. MacW. and G. S. M.)."

"The auditory phenomena were very marked and characteristic, the phases corresponding with those found in man and in a circulatory schema. The systolic auditory index was found to be correct. The auditory diastolic index constituted by a sudden dulling and weakening sound at a certain point in the lowering of the armlet pressure proved a very accurate guide to the intra-arterial diastolic pressure as shown by the minimum manometer.[1] Extinction of sound was much later."

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[1] "We arrived at this conclusion on other grounds also, from experiments with a circulatory schema as described in *Heart*, 1914, p.193, and from observations made on man in different circumstances; see paper in the *Quart. Journ. of Exp. Physiol.*, 1914, vol. viii, and one in the *British Med. Journ.*, March 1914."

"Considerable variations in pressure and pulse rate were induced in the course of the observations with no impairment of the accuracy of the results. The pressure was raised by mechanical compression of the abdomen, lowered by an increased dose of chloroform, etc. Pulse rates varying from about 60 to 110 were present at the different phases."

"We also tried the oscillatory method by connecting the armlet with the Erlanger sphygmomanometer and with the Pachon sphygmo-oscillometer, but found the indications more difficult to read than the auditory ones."

"At the beginning of the observations before the armlet was inflated, the pressures, as shown by the manometers, were: Systolic 110, Mean 80, Diastolic 52, the mean pressure being only slightly different from the arithmetical mean."

Some experimental data are subjoined:

	Intra-art. Pressure by Valved Manometers.			
Observation.	Systolic.	Diastolic.	Auditory Diastolic Reading.	Remarks.
1	96	45	45	Much chloroform given.
2	96	51	50	
3	98	54	50	
4	96	56	53	
5	96	60	60	
6	102	84	80	Compression of abdomen. Pulse rate 110.
7	96	60	60	

	Intra-art. Pressure by Valved Manometers.			
Observation.	Systolic.	Diastolic.	Auditory Diastolic Reading.	Remarks.
8	112	70	70	Compression of abdomen.
9	112	70	70	
10	85	41	40	Dose of chloroform increased.
11	85	48	47	
12	94	46	45	
13	94	74	74	
14	94	65	68	
15	80	56	54	

This verification of the diastolic criterion at the point at which the clear note of the third phase ceases and the dull note of the fourth begins is a matter of considerable practical importance; for G. S. Melvin and J. R. Murray have found that the duration of the fourth phase is not a question of some 5 mm. only, as has been hitherto held,[1] but may in any particular case extend from a few mm. to 30 or even 55 mm., and these observers remark: "It is clear from our results that the persistence of the fourth phase may often be so long that to take the lower limit as the diastolic index would, in many cases, lead to hopelessly erroneous results." "It is to be noted that the average duration of the fourth phase is vastly greater than has been suggested by former observers, and that there is no constant association with variations in pulse rates, systolic pressures, or pulse pressures." The clinical significance, if any, of the length of the fourth phase is unknown.[1]

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[1] I have hitherto regained 5 mm. as the average length of the fourth phase. "Goodman and A. A. Howell state 6 mm. Warfield puts it, for normal pressures, at 4 to 10 mm., occasionally not more than 2 to 4 mm., but with high pressures never less than 8, and sometimes as high as 16 mm. Weysse and Lutz make it at most 25 mm." (quoted from the paper "Diastolic Blood-Pressure Estimations by the Auscultatory and Oscillation Methods," by G. S. Melvin and J. R. Murray in *Quart. Journ. of Experimental Physiology*, 1914, vol. viii, p. 129).

The Insufficiencies and Advantages of the Auditory Method.—My observations with the auditory method enable me to endorse the following summary of the conclusions formed by J. A. MacWilliam and his co-workers as to the limitations and advantages of that method—founded as they are on well-attested experimental and clinical data.

Limitations and Sources of Fallacy are Few and Easily Guarded Against.—"(1) In many cases of aortic insufficiency the well-known sound (systolic thud or shock and sometimes the double murmur of Duroziez) make the auditory method inapplicable.

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[1] In the majority of hospital cases and of middle-aged and elderly persons and in young children the fourth phase is very short, and in subjects with thickened arteries and high arterial pressure it is specially so or almost absent, but in young adults (men and women) it is frequently considerably extended (communicated by Prof. MacWilliam).

"(2) An obstructed flow of blood through the brachial artery impairs the validity of the auditory criteria. This may arise from congestion of blood in the limb distal to the armlet from unnecessarily prolonged constriction,[1] or from an excessively small calibre of the brachial artery or its continuations in the forearm.

"(3) An extremely low pulse pressure, due to small cardiac output (feeble heart, very excessive pulse rate, etc.), with relaxed peripheral vessels.[2]

"(4) While the brachial artery is excellently adapted for this method, application of the armlet to the forearm gives unsatisfactory results as a rule. The range of sound heard over the radial artery is defective, being shortened both above and below, *i.e.* the systolic index is too low, and the diastolic too high.

"(5) The possibility of conduction of the sound along a bone occasionally comes into question both as regards the systolic and the diastolic index.

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[1] Prolonged constriction and congestion of the limb is met by adopting one or other of the rapid methods of filling described on pp. 8, 11, and 138.

[2] In such cases, and sometimes in the obese in whom the throbs are sometimes feeble from deposition of fat, the observer should extend the elbow, so as to project the artery; and if this fails to furnish the full range of throb, he should rely on the tactile index for the systolic and on Pachon's visual index afforded by my manometer with the 3 mm. bore for the diastolic reading (see p. 5).

"(6) Improper adjustment of the auditory tambour might possibly lead to inaccuracy, but not when the tambour is properly situated with no appreciable pressure.

"In all the conditions mentioned, insufficiency of flow is the leading source of fallacy ; the sound on decompression being delayed—thus affording an under-estimate of the actual systolic pressure—and declining too soon—thus making the diastolic reading too high. But such an undue lowering of the upper limit of sound is readily recognisable by using the tactile systolic index simultaneously, and this affords a most important indication of the unreliability of the sudden declension as an index of diastolic pressure. We adopt and recommend as the routine procedure the simultaneous determination of (1) the reappearance of the pulse by the ordinary tactile method, and (2) the auditory systolic index as a method of verifying the correct arrangement of the auditory tambour, and its applicability to the conditions present in any particular case."

The Advantages.—(1) The simplicity and quickness of the procedure are obviously very great considerations, dispensing, as it does, with relatively cumbrous and time-wasting apparatus (smoked drum, recording tambour, etc.).

(2) The avoidance of the discomfort and reflex disturbance of the whole circulation caused by the prolonged and uninterrupted constriction and congestion of the limb, necessitated by the oscillatory method in pushing up the armlet pressure well above the obliteration point, and in slow decompression, either for the graphic record or for the repeated observation of the range of oscillation at frequent stages in the visual method of Pachon.

(3) The much greater definiteness of the systolic and diastolic index, as compared with the difficulty and uncertainty of that of the oscillatory method (Recklinghausen—Erlanger and Pachon).

(4) The greater constancy of the results. Apart from movements of the limb, the Erlanger record is apt to vary much, and this is not surprising in view of the complexity of the conditions which may be present, and play a part in influencing the oscillations. The auditory method is much less disturbed by alterations in the arterial walls (resistance, distensibility, etc.); this naturally results from the required degree of flattening of the arterial tube being very much less than in the other (oscillatory) method, while, in a vessel presenting unequal resistance along its course, change from the circular form in a comparatively short piece suffices to produce a sound, while flattening along the whole or

the greater part is needed to give maximal oscillations; numerous influences may tend to give a gradation in the development of the oscillation, and an absence of any sudden and striking change in the size of the oscillations. The greater degree of flattening approaching complete flattening necessary to give maximal oscillation in a practically non-distensible tube is an important factor, while the form of the pulse curve may also be such as to aid in increasing the excess of external pressure required. The varying influence of volume changes due to elongation does not come into question in the auditory method.

(5) The auditory diastolic reading is quick and simple, more precise and easily interpreted, and is less disturbed by abnormal conditions than that yielded by other methods (visual, graphic, etc.), rendering the latter unnecessary. It is a procedure that takes very little time and trouble, and yields very definite and valuable information; moreover, it produces less circulatory disturbance in abnormally sensitive subjects than the stronger (obliterative) constriction of a comparatively large vascular area involved in systolic determination; and is much less, if to any appreciable extent at all, affected by the main sources of fallacy in systolic estimation, such as the resistance of the arterial wall, possi-

bility of reflected waves, etc. Even comparatively slight changes in diastolic pressure, unless dependent on alterations in the pulse rate, are of considerable significance as indications of circulatory conditions (changes in peripheral resistance, etc.).

(6) The auditory systolic reading should also be made as a routine practice, and this should be checked by the ordinary tactile method.[1]

(7) The "pulse-pressure range" (see p. 43) as determined by the auditory method is much more extensive in many pathological conditions than is indicated by the ordinary accepted ratios.

(8) It is clear that the auditory method must supersede the maximum oscillation method and others that have been proposed—based on the recognition of changes in the artery distally to the armlet; the last named we have found to be unreliable. The maximum oscillation method in its best form is relatively elaborate and cumbrous, subject to many causes of variation, dubious in its indications in many cases, and quite unnecessary in view of the simplicity and reliability of the auditory method.

In a recent study of "Blood-Pressure Estimation in Disease, by the Oscillatory and Auditory Methods," G. Spencer Melvin and J. R. Murray disclose a marked discordance between the two methods of reading the diastolic pressure and the range of pulse pressure in fifty-one pathological cases, as compared with the practical concordance in a series (fifty-nine subjects) of normal young adults; and they conclude their study with the following comment: "In the presence of such discordance, we prefer the auditory readings, in view of the evidence available as to the complexity and variability of the oscillation method in different conditions, and the reliability of the auditory indications as verified by comparison with direct measurement of the internal pressures, by valved manometers in an experimental schema and in animals; in both of these the auditory index was found to be a remarkably accurate guide to the actual diastolic pressure."[1]

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[1] See also *Pro. Roy. Soc. Med.*, 1910, vol. iii (med. sec.), p. 207.

IV. Normal and Pathological Readings of the Arterial Pressure

Normal Readings.—In the following table the average systolic, diastolic, and pulse pressures are arranged for the leading stages of life, represented by children, young adults, the middle aged, the elderly, and the aged. The outside limits of normal variation (plus or minus) are not given; but for practical purposes these will be covered by adding to or subtracting from the average figures 15 mm. for the systolic, and 5 to 7 mm. for the diastolic pressure readings.

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[1] *Quarterly Journ. of Medicine*, 1914, vol. vii, pp. 419–26.

I prefer this mode of estimating the ordinary physiological limits of variation of the arterial blood-pressure to that usually adopted; because it enables us to make a better estimate of the pathological bearings of the arterial pressure at different ages than we can obtain by fixing for all ages the upper limit of 150 or 160 mm., and the lower limit somewhere short of 100 mm. This upper margin of normal pressure is useful enough when the patient has passed his sixtieth year or so; but is misleading in younger subjects, and especially in those below forty.

Average Normal Arterial Pressures determined by the Auditory Method with Tactile Systolic Check.

Age.	Systolic Pr.	Diastolic Pr.[1]	Pulse Pressure.
Under 15[2] . .	107	74	33
20–40 . . .	112–125	65–80	45
40–60 . . .	125–135	80–85	45–50
60–80 . . .	135–165	85–90	50–75
Over 80[3] . .	Reduction of all the pressures		

[1] All previous estimates of the normal diastolic pressure determined by the maximum oscillation criterion are too high.

[2] Data supplied by G. Spencer Melvin & J. R. Murray, *Quarterly Journ. of Exper. Physiol.*, vol. viii, p. 136.

[3] Data insufficient.

F. A. Faught's rule for calculating the normal average systolic pressure is very simple and easily kept in mind; consider it to be 120 mm. for a male subject of twenty, then add 1 mm. for every additional two years of life.[1]

The best series of normal systolic pressures on record is that afforded by 13,067 lives accepted by the North-western Mutual Life Insurance Co.[2] from 1907 to 1911.[3] The figures are arranged in quinquennial stages.

Ages.	Number of Cases.	Average Systolic Pressure.[4]
15–19	21	118·00
20–24	56	123·53
25–29	80	125·61
30–34	86	125·50
35–39	177	126·25
40–44	5,424	128·26
45–49	3,867	130·51
50–54	2,169	131·98
55–60	1,187	134·46

Pathological Readings.—In deciding on the normality or abnormality of any individual arterial blood-pressure we should take into consideration several data: such as (1) the average maximum above or the average minimum below the general average reading for age in a large group of healthy subjects; (2) the collateral clinical evidence in the patient of the cardio-vascular syndromes of hypertension and hypotension and of the pathological causes which raise or lower the arterial pressure; and (3) the conditions furnished by the individual which may modify the readings of arterial pressure.

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[1] *Blood-Pressure from a Clinical Standpoint*, by F. A. Faught, M.D., etc., Philad. and Lond., 1913, p. 221.

[2] This insurance company does not accept lives over sixty.

[3] *The Diagnostic Value of the Use of the Sphygmomanometer in Examinations for Life Insurance*, by J. W. Fisher, M.D., Medical Director of the North-western Mutual Life Insurance Co., 1911.

[4] The auditory reading of these figures would be a little higher (3 to 5 mm.).

H. P. Woley[1] has recorded and averaged the low and high readings of the systolic pressure in 1,000 healthy subjects from the age of fifteen to sixty-five, observing all possible precautions.[2] The following table is an epitome of his results:

	Readings of Systolic Pressure.[3]		
Age.	Average Low.	Average.	Average High.
15–30	103	122	141
30–40	107	127	143
40–50	113	130	146
50–60	115	132	149
(55–65	120	138	153)

The averages for females are 7·5 lower.

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[1] *Journ. of American Med. Assoc.*, 1910, vol. lv, pp. 121–3.

[2] Uniform standard conditions in armlet (12 cm. broad), posture (sitting) were observed and the results were frequently checked by two physicians working together.

[3] The auditory readings of the figures would be from 3 to 5 mm. higher.

If we place the commencement of pathological readings 5 mm. or so outside these high and low averages of the physiological systolic pressure, we obtain figures which practically conform with the pathological ones furnished by general clinical experience; namely, above 145 or below 100 before middle life (forty), or over 160 or under 110 after. But of course such figures must be verified by repetition, not only on one, but on more than one occasion, and by the exclusion of avoidable fallacies (see Chap. V.). In the above table the high and low figures for age in the apparently healthy therefore border on the readings of hypertension and hypotension, and they are clinically important; for they should suggest more particular inquiry, so that preventive treatment may be adopted early when that is found to be necessary.

Pathological systolic readings in hypertension may extend from 145 to 160, according to age, to at least 300 mm.; and in hypotension they may fall from 100 or 110 to 60 mm., or even lower. In hypertension, readings of 200–220 are moderately high; of 250–275 are very high; and of 280–300 are not only excessively high, but are quite exceptional.

The range of pathological diastolic readings is about 50 per cent. less than that of the systolic; and the individual differences observed within the same decade are also diminished to the same extent. Therefore we should attach clinical significance to comparatively small increments and decrements of the diastolic pressure. Diastolic readings of 90–100 in subjects under forty and of 100–110 after that age, when verified on more than one occasion, should call for precautionary inquiry; and those of 120–130 are moderately high, of 150–160 are very high, and of 165–175 are excessively high.

The measurement of the pulse pressure (see p. 43) and of the brachio-digital difference (see p. 59) may provide corroborative or corrective evidence of the normality or abnormality of the arterial blood-pressure.

Qualifying Conditions.—In deciding whether any particular arterial pressure is pathological or not, the observer should not be altogether bound by figures which denote the outside limits of normal pressure for age; he should take into consideration certain qualifying conditions such as sex, temperament, mode of life, body weight and build, and heredity.

In women, the systolic pressure and pulse pressure are, as a rule, about 10 mm. and the diastolic 5 mm. less than in men.

Nervous, anxious subjects, and occupations which involve much anxiety, worry, and nerve-strain, yield somewhat higher levels of arterial pressure —especially in the latter half of life. On the other hand placid temperaments and routine occupations, especially of the physical kind, dispose to the lower degrees of pressure. In subjects in good condition and training, such as athletes, the arterial pressure is very often below, rather than above, the normal average; a fact which points to the importance of maintaining throughout life the functional activity of the peripheral circulation by exercise. And this inference is supported by the observation that in those normal subjects who follow sedentary indoor occupations for many hours daily, the pressure, though generally normal, is more frequently above and less frequently below the normal pressure-line for age.

Small, light subjects, especially women, have generally somewhat low arterial pressures ; so that in such cases a rise above the normal for age, which may be comparatively slight, should be regarded as more significant than in subjects of average build and weight. Broad or largely built subjects, especially when obese, often yield higher arterial pressures, which for them are generally not abnormal ; and when in such cases the pressure is subnormal, there may exist clinical indications of its inefficiency.

In certain families, heredity plays a part in inducing higher or lower ranges of arterial pressure.

The modifying influence of unusually hot or cold weather (the hypotonic effect of heat and the hypertonic effect of cold) on the blood-pressure should also be kept in mind. According to my observation, warm weather, especially when decided and continued, may let down the systolic blood-pressure from 10 to even 20 mm., and cold weather may raise it to a similar extent. Hence during spells of hot weather an arterial pressure may appear to be quite normal, whereas when the temperature is ordinary and temperate it may exceed the upper physiological limit; and in very cold weather the pressure may seem to be higher than it is at other times.

There is also some evidence to show that in tropical countries (such as India) the arterial pressure is lower than in colder and more temperate countries. McCay of Calcutta thus writes: "The pressure (taken *à la* Riva-Rocci) over a very large number of observations varied between 83 mm. Hg. and 118 mm. Hg., the average being slightly over 100 mm. Hg. (sitting position, arm level with heart). This is much lower than the blood-pressure laid down for Europeans in European (colder) countries."[1]

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[1] *Lancet*, vol. i, 1907, p. 1484.

CHAPTER V

SOURCES OF FALLACY

THE measurement of the arterial blood-pressure is open to certain fallacies, which a little experience generally enables the observer to modify, or even to obviate. There are two principal sources of error, which produce an apparent rise in the pressure: namely (1) some temporary stimulation of the cardio-vascular system, and (2) the state of the arterial wall.

I. TEMPORARY DISTURBANCE OF THE ARTERIAL BLOOD-PRESSURE

The aim of the observer is to discover some more or less continuous variation of the arterial blood-pressure. He must therefore be always alive to the possibility that the particular pressure under observation is merely temporary. Such passing increments of pressure are generally due to excitement, and are always associated with an increased pulse rate; as a rule, however, they quickly subside as soon as the patient becomes familiarised with the new and unexpected procedure, and especially after the observer has let fall a few reassuring words. But while the majority of patients are thus tranquillised, there is a small minority in whom nervous sensibility is a disturbing element of more or less difficulty. Some of these cases are so sensitive as to be disturbed by any examination of the pulse —even counting its rate; while others are more particularly disconcerted by the constriction of the armlet—especially when the readings exceed 180–200 mm. Hg. and by the painful sense of distension in the limb. But now the latter difficulty is cut down considerably, or even practically cut out, by the method of rapid filling (see p. 138). Still, there remain a certain modicum of cases intrinsically unsuited for trustworthy measurement of the arterial blood-pressure.

II. The State of the Arterial Wall

As "the soft tissues of the arm are practically negligible as a source of error,"[1] whatever anatomical source of fallacy exists in determining the arterial blood-pressure in man must rest with the arterial wall. The solution of this question has been approached either from the side of experiment with arteries in various conditions (dead, surviving, contracted, relaxed, or thickened) in circulatory schemata, or from that of clinical observation of the pressure in the arm and leg, or in different portions of the limb, *e.g.* arm and forearm, thigh and calf, in which, in the horizontal position, it should be identical. To describe the points of technique adopted, and the detailed results observed by the various experimenters in their inquiries, would be out of place in this practical work. I shall therefore merely quote their conclusions. Trustworthy clinical evidence does show that decided differences in the readings of the arterial pressure taken simultaneously, or in quick succession, in the arms, or in the arm and leg, are every now and then met with. It is conceivable that such differences may be due to (1) altered conduction of the pressure of the pulse-wave, (2) variations in the resistance of the arterial wall to compression, and (3) reflection of pressure from the periphery. The last-named cause of variation may, however, be dismissed as problematical.

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[1] *Archives of Intern. Med.*, 1909, vol. iii, p. 474, "The Influence of the Soft Tissues of the Arm on Clinical Blood-Pressure Determinations," by T. C. Janeway, M.D., M.A., etc. The presence of œdema (rare) and of much fat in the arm is, however, unfavourable for the accurate reading of the arterial pressure.

The experimental studies of Leonard Hill,[1]

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[1] *Journ. of Physiol.*, vol. xxxviii (Proc. Physiol. Soc.), p. 48, Hill

Flack, Holtzmann, Rowland, and Russell Wells lead these observers to attach great importance to the influence of the arterial wall in modifying the conduction of the aortic pressure; believing that much more of this pressure is lost in dilating arteries, which are soft and flaccid, than in those which are thickened and rendered less resilient from muscular contraction, or from organic changes. Hence on this assumption the same aortic pressure will yield a lower reading of the brachial pressure in the former condition of the arterial wall than in the latter. These observers also believe that the large excess (sometimes over 100 mm.) of the leg readings in cases of aortic regurgitation, over those of the arm, is caused by contraction and rigidity of the leg arteries, which conduct the aortic pressure better than the more resilient brachial artery.

At first I accepted this interpretation as probable;[1] but after studying the experimental evidence of J. A. MacWilliam,[2] J. E. Kesson, and G.

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and Flack; *Heart*, 1911–12, p. 219, Hill and Rowland; *ibid.* 1908–9, vol. i, p. 72, Hill, Flack and Holtzmann; *Proc. Roy. Soc.*, Series B, vol. lxxxvi, 1913, p. 180, Hill and Russell Wells.

[1] *Practitioner*, 1914, p. 91.

[2] *Heart*, vol. iv, 1913, pp. 393–408, "The Conduction of the Pulse-Wave and its Relation to the Estimation of Systolic Pressure," by J. A. MacWilliam, J. E. Kesson, and G. Spencer Melvin.

Spencer Melvin I can no longer do so. These observers remark: "If this view is correct, the leg readings would afford more accurate indications of aortic pressure than the arm readings, the aortic pressure really being vastly higher than is indicated in the arm. Such a hypothesis would involve the occurrence of a very great loss in transmission between the aorta and the brachial as much as 80 mm., 100 mm., or more in some cases;" and they conclude their article thus:

"While the elasticity of the aorta and its branches is of great importance in regard to the systolic and diastolic pressures in the arterial tree—giving lower systolic and higher diastolic pressures than would otherwise be present—the transmission of the systolic pressure from the aorta to the brachial artery is not, under the ordinary conditions of blood-pressure estimation by the obliteration method, appreciably influenced by the resilience or rigidity of the intervening arterial tube.

"The difference sometimes observed between arm and leg readings between the two arms, arm and forearm, etc., are not to be explained by differences in the conduction of the systolic wave from the aorta depending on the character of the arterial tubes in the different limbs, etc. Diminution in the calibre of an artery, if extreme, may influence the propagation of the systolic wave, but under the ordinary conditions of blood-pressure estimation in the arm, this factor is a negligible one."

There remains for consideration the question of the varying resistance in the arterial wall, as the explanation of the observed differences in the readings of the arterial pressure. On this point I shall quote the conclusions formulated by two sets of laboratory observers, who are not only well equipped as experimentalists, but thoroughly conversant with the clinical side of sphygmomanometry. Their inquiries, moreover, being of recent date, are free from errors in technique which impaired the validity of previous investigations. The research of Theodore C. Janeway and Edwards A. Park[1] yields the following conclusions:

"1. The arterial wall offers definite resistance to compression.

"2. Other things being equal, small arteries with thin walls are more readily compressed than large arteries with thick walls.

"3. In infancy and childhood the resistance of the arterial wall is a negligible factor in clinical blood-pressure measurements.

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[1] *Archives of Int. Medicine*, 1910, vol. vi, pp. 586–613, "An Experimental Study of the Resistance to Compression of the Arterial Wall," by Theodore C. Janeway, M.D., and Edwards A. Park, M.D.

"4. Arteries as large as the brachial may require only the pressure of a few millimeters of mercury for the obliteration of their lumen.

"5. In adults with normal arteries and a normal range of blood-pressure, the arterial wall is a practically negligible factor. It probably never introduces an error greater than 10 mm. Hg. in clinical blood-pressure measurements, a figure less than the spontaneous variations in pressure from minute to minute.

"6. Atheroma, even of considerable degree, is without appreciable effect on the compressibility.

"7. Calcification of the arterial wall, when segments longer than 6 cm. are examined, increase only moderately its resistance to compression. The over-pressure dependent of this factor in our experiments did not exceed 17 mm. Hg.[1]

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[1] The experimenters remark: "It is evident that the average pressure required to compress these (calcified) vessels was 5·5 mm. above the average for the slightly atheromatous post-mortem vessels. The minimum was strikingly higher, 10 mm., while the maximum was actually 2 mm. below the highest reading from an artery with almost normal wall. It is, therefore, against evidence that even calcification of the most extreme degree has less influence on the compressibility of the arterial wall than some other factor or factors. From our observation of the behaviour of these arteries; we think it probable that no such vessels will be found calcified throughout their whole circumference, for any considerable distance. It is altogether unlikely, therefore, that any strip of such a vessel, as much as 10 cm. long, should fail to have soft or uncalcified spots

"8. In clinical blood-pressure determinations, if a wide armpiece be used, and the return of the first fully developed pulse-wave be taken as the index, as recommended by Recklinghausen, even advanced arterial thickening and calcification probably do not introduce an error of any importance.

"9. The only factor determining the compressibility of an artery, which seems capable of introducing an error of real importance in the clinical measurement of systolic blood-pressure, is the state of contraction of its walls. It is impossible from our experiments on surviving ox arteries to set definite limits for this in man. From these experiments, however, combined with our study of human arteries, after amputation and post-mortem, we feel that a degree of hypertonic contraction of the brachial artery, sufficient to cause an error of more than 30 mm. Hg., seems improbable, and more than 60 mm. incredible, during life.

"10. The point of return of the pulse after ob-

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in its walls. Such soft spots may be easily compressed against a calcareous plaque on the opposite side of the vessel. The inspection of these vessels, while undergoing compression, has shown collapse of the vessel in a small area, while the rest still stood open, and has convinced us that the foregoing explanation accounts for the apparent disparity between their behaviour under the finger and in our apparatus, thus the need for the broad armpiece in clinical work is emphasised from a new standpoint."

literation, not of its disappearance during compression, should always be the criterion of systolic blood-pressure."

The investigation of J. A. MacWilliam, J. E. Kesson, and G. Spencer Melvin[1] on the influence of the arterial wall in blood-pressure measurement is based on broader lines than those of previous workers. It traces that influence on the oscillatory and auditory methods of reading the diastolic, as well as the systolic, determinations; and it embraces a clinical study of the arterial blood-pressure by the light of experimental data. These data were derived from the examination of long lengths (10 to 16 cm.) of excised living[2] arteries (human and animal, relaxed and contracted, normal and altered by old age and disease) subjected to internal pressure (pulsatile and non-pulsatile) and to external pressure in a compression tube connected with three valved manometers (maximum for the systolic, minimum for the diastolic, and "compensated" for the mean pressure). The circulation through the arteries, adjusted to a dummy humerus, was likewise subjected to the armlet pressure.

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[1] *Heart*, vol. iv, 1914, pp. 279–318, "The Estimation of Systolic Blood-Pressure in Man, with special reference to the influence of the arterial wall," by J. A. MacWilliam and J. E. Kesson. *Ibid.*, pp. 133–96, "The Estimation of the Diastolic Blood-Pressure in Man," by J. A. MacWilliam and G. Spencer Melvin.

[2] Dead arteries and rubber tubes afford misleading results.

The experiments on excised surviving arteries afford the following results :

"(1) The relaxed arteries, whether normal or thickened,[1] gave very inconsiderable resistances to compression, not usually more than a few mm. of Hg.

"(2) Contracted arteries showed resistances varying with the amount of contraction present. Arteries powerfully contracted at room temperature may give high values, rising sometimes to over 100 mm. Hg., in the metacarpal or metatarsal artery of the horse, and in the carotid of the ox. Arteries contracted at body temperature yield much lower figures, a maximum reading of 35 mm. for the carotid of the sheep, 64 mm. for that of the ox, and 60 mm. for the metacarpal of the horse. These are maximum values obtained at the first compression of the pulse disappearance index; with the pulse re-appearance at the second compression, the values are much lower. Arteries offering great resistance when strongly contracted show very little when fully relaxed.

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[1] "The great bulk of available evidence is opposed to the idea of thickened arteries offering any important resistance, apart from the presence of muscular contraction ; this is in agreement with the results of von Basch, Martin, Janeway, and Park, and others working with dead sclerosed arteries. The only positive piece of evidence in the contrary sense is that advanced by Herringham ; his results were obtained from dead arteries."—J. A. MacWilliam and J. E. Kesson, *Heart*, vol. iv, p. 296.

"(3) Arteries contracted and resistant to external pressure were found to be quite compressible under nominal pressures (*e.g.* 1 mm. Hg.) when a very limited local softening (*e.g.* 3–5 mm. in width) was produced by pinching the wall between the blades of an artery forceps; and to be again highly resistant when the softened area was protected from compression by a short piece of rigid tubing slipped over it. These very definite and convincing results make it plain that the inflated bag of the armlet acts effectively on a very limited soft portion of a thickened and resistant artery, and prevents the resistance offered by the rest of the vessel from affecting the obliteration readings much.[1]

"(4) When contraction causes extreme constriction of the arterial lumen below a certain point, the transmission of the systolic wave to the seat of compression may be diminished, and this factor may lessen the effect of the increased resistance of the arterial wall upon the obliteration readings in arteries capable of having their lumen reduced to a minute size. Thickened metacarpal and metatarsal arteries from old horses often contract so much as to close their lumen completely, and resist any passage of fluid through them, even at pressures up to 440 mm. Hg.

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[1] These observations are in line with these of Theodore C. Janeway and Edwards A. Park, on the behaviour of soft areas in calcified arteries favouring their early occlusion under compression.

"(5) Repeated compression or continued local compression, while not affecting the readings from normal arteries under ordinary conditions, has an important effect in reducing the resistance of a contracted artery, and may be used as a means of ascertaining the presence or absence of abnormal resistance in the arterial wall. Massage of a resistant artery may be used with similar effect."

The following are the leading conclusions furnished by the clinical inquiry:

"(1) Estimations of the systolic blood-pressure by the obliteration method, when made with suitable precautions, give substantially correct results in ordinary conditions of normal health, and also in the great majority of cases of illness, in the collective sense. Even when the disease affects the vascular system with thickened arteries, etc., the indications are, in the majority of cases, approximately correct; the readings ranging from moderate or low, to very high values. It is only in a minority of cases that any serious error is liable to occur, in the direction of over-estimation.

“(2) In some such cases the influences of local conditions may be very important, especially the presence of abnormal resistance in the arterial wall, depending, mainly at least, on contraction of the muscular coat. In such conditions very different readings may be obtained from the same person on the same occasion, according to the limbs, or parts of limbs, examined, the using of first[1] or later readings, etc.

“(3) Continued and repeated compression, with comparison of the two sides, etc., affords a valuable method of detecting the presence of such error, though not invariably decisive; in some instances considerable disturbances of blood-pressure may occur. Results, both positive and negative, obtained by this method, show a striking parallelism to those yielded by excised surviving arteries.”

How to Relax and Soften Contracted and Otherwise Thickened Brachial Arteries for Blood-Pressure Observation.—An important practical outcome of this investigation is the discovery of a method by which rigid arteries may be softened. It having been proved that repeated compression or massage of resistant excised surviving arteries in the compression tube increases their compressibility very considerably, their resistance in some cases being cut down to practically the value of the relaxed wall, the observers applied this method to various cases presenting a normal or abnormal condition of the arteries in the arms or forearms. An armlet was adjusted to each arm (or to each forearm, or to arm and forearm) and both were connected with a three-way tap through which they could be inflated simultaneously or in quick succession;[1] then after making a synchronous estimation in the two arms, repeated compression was applied to one arm, the one giving the higher reading in cases of initial inequality, the pressure being let down to zero between each obliteration. Another synchronous estimation in both arms was then made to find the effects of repeated compression in one, and to detect possible changes in the general blood-pressure by the reading from the other arm. In cases in which the circulatory balance was easily disturbed, obliteration of one artery at a time was preferred. Instead of repeated compression by successive inflations of the armlet, digital obliteration of the brachial in mid-arm was kept up for two or three minutes. The observers thus classify the effects of continued or repeated compression applied clinically:

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[1] The first reading may be a most fallacious one.

"(1) In the generality of cases, when no abnormality of the vascular mechanism is present, the result is practically nil, excepting that in some persons there may be a decided reduction of the obliteration reading not dependent on an alteration in the resistance of the arterial wall, but due to the passing off of slight alarm, excitement, etc., which may be present at the first reading. That such is the cause of the reduction is indicated by a reading from the other limb, in which the first reading gives the same lowered level as is found in the first limb, after repeated compression.

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[1] In the latter case a tap is fixed for alternate inflation.

"(2) Similar negative results are obtained in many cases of arterial disease where the thickening is of an uneven or patchy character, or associated with atrophic changes in the media and hypertrophy of the intima, atheroma, etc., or where the hypertonus is unevenly distributed, as is often seen in excised arteries, when high readings are obtained, not appreciably lowered by repeated compression; the diastolic readings are also frequently high, though by no means always, in the absence of abnormally slow heart-beat, aortic regurgitation, etc. Excessively high readings, not reduced by the method in question, are found both with soft arteries not recognisably thickened, and also with thickened and extremely tortuous ones that feel resistant to the finger.

"(3) In many cases where an abnormal resistance is presented by the arterial wall from muscular contraction, with or without thickening of the media, repeated compression produces a marked lowering of the readings, essentially due to a diminution of the resistance, though it may at times be associated with some lowering of the general blood-pressure, as indicated by the reading of the other arm, etc. Such reduction is a valuable indication of the presence of abnormal resistance in the arterial wall, a factor which makes the initial reading yield an exaggerated estimate of the actual blood-pressure within the vessel. A very high initial reading falling, on repeated compression, to a moderate level is commonly associated with a moderate diastolic pressure, excessively small in proportion to the initial (exaggerated) systolic reading. The reduction may be very unequally manifested in different limbs, or segments of limbs. Very notable differences (50 to 90 mm.) are sometimes found between the two arms, between arm and forearm, and between arm and leg. These differences are not necessarily constant; they may be found to alter markedly when examined at intervals of a few days, or during the process of examination by repeated compression. The latter method commonly reduces the difference, if it does not remove it. In some instances the difference found in the two arms at the first reading has remained practically constant over long periods (many months). We have found (though rarely) arm differences so great as to be comparable to the arm-leg differences in obliteration values that have been described in aortic regurgitation.

"(4) The first reading, whether disappearance or (preferably) re-appearance of the pulse is taken as the index, in cases with normal arteries, or pathological arteries, presenting no appreciable or important resistance to compression, is approximately correct as an indication of the actual systolic pressure. The actual pressure may, however, be influenced by excitement, etc. When there is marked resistance in the arterial wall, a first reading may give very seriously erroneous values. Repeated compression greatly reduces or rectifies this error in many cases. The number of recompressions necessary to do this varies greatly.

"(5) In some cases with abnormal instability of the vascular mechanism a more or less extensive rise in the readings is produced by recompression, depending, mainly at least, on a rise in the arterial blood-pressure. Such rise in general blood-pressure may more or less completely conceal, or may quite overbear, the effect of any reducing influence exercised upon the resistance of the wall of the compressed artery; and consequently we cannot, by the obliteration method, obtain an accurate estimate of the arterial blood-pressure in such cases."

This investigation corroborates to a large extent those previous clinical observations which pointed to the arterial wall as the source of disparity in systolic pressure readings in different areas; such as those of O. K. Williamson[1] and my own.[2] But it indicates that the cause of that disparity is not so much to be ascribed to organic differences in the thickness and resistance of the arterial wall as to muscular contraction of the tunica media; and therefore it supports to some extent the views of W. Russell[3] on the disturbing influence of hypertonus on blood-pressure readings.

The inquiry also confirms the experience of those who always reject the early readings of the systolic pressure, and prefer to wait until the reading remains steady and at its lowest point; for in following this rule they really employ in a modified way the method of repeated compression of the artery.

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[1] *Proc. Roy. Soc. Med.* (med. sec.), 1908–9, vol. ii, p. 229.

[2] *This work*, 2nd ed. 1908, pp. 111–22.

[3] *Arterial Hypertonus, Sclerosis and Blood-Pressure*, 1908.

CHAPTER VI

TECHNIQUE

The Patient.—No trustworthy observation of arterial blood-pressure can be made without quiescence in the patient—quiescence mental, emotional, and physical.

After exertion, sufficient time should elapse to allow the pulse rate to subside to normal. With many sensitive subjects, the observation should be delayed until the patient has become quite settled down from any apprehension incidental to the medical examination of his case. This precaution is all the more necessary should the pulse be hurried from emotional excitement. The observer should lead up to the unaccustomed method of observing the pulse, and should avoid surprising the patient by raising the armlet-pressure before telling him what he is about to do.

The observation can be made in the sitting or recumbent posture. In the former, which is more convenient and expeditious for clinical work generally, the arm should rest comfortably, with all its muscles relaxed, on a table of convenient height, so as to bring the armlet on a level with the heart. In the recumbent position, the arm should be extended on a table or other support, by the side of the bed or couch, and on the level of the body. The armlet in both positions occupies much the same gravity position in relation to the heart. The arm should be bared ; though a thin covering under the armlet does not alter the reading of the pressure, it does impair the sensitiveness of the phonendoscope, which should be adjusted under it, at the bend of the elbow.

The Position of Armlet.—The armlet may be adjusted to either the arm or the forearm ; and each of these possesses certain advantages and disadvantages. The arm, from its cylindrical form, even compressibility, and its single bone and large artery, is anatomically better adapted to hæmomanetric observation than the forearm with its smaller arteries. And for the employment of the auditory method of reading the pressure, the arm is the only part available ; for though that method may in some cases yield satisfactory results when employed on the forearm, in others the range and calibre of the throb are curtailed. The principal objection to the use of the armlet on the arm is, however, the larger amount of discomfort from congestion of the limb than obtains with the armlet on the forearm; but this is met by the quick methods of compression and decompression about to be described. The outcome of these considerations points to the arm as a better position for the armlet than the forearm. The latter is, however, sometimes more convenient, especially in the case of ladies, when the observer should employ the tactile method for the systolic, and the oscillatory for the diastolic pressure. But in making observation on the forearm the observer should bear in mind that he may, in exceptional cases, make an under-estimate of the arterial blood-pressure, because the smaller calibre of the arteries (radial and ulnar) is more apt than the brachial to be so reduced by contraction as to lower the blood-pressure within them.[1]

The air bag of the armlet should be placed along the course of the brachial artery, or on the flexor side of the upper half of the forearm, when this position is more convenient, and the straps should be adjusted comfortably and closely.

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[1] In the case of children the armlet can be applied either to the forearm, which provides definite auditory readings of the systolic and diastolic pressures, or to the thigh, when the tactile systolic criterion can be read on the posterior tibial artery, and the auditory criteria (systolic and diastolic) by applying the phonendoscope over the popliteal artery. A narrower armlet is more convenient for the forearm.

Objections to the Armlet Pressure.—Those who have had some experience in the use of the armlet among private patients soon discover that it is not faultless as a clinical method. From the patient's standpoint, the necessary compression is, as a rule, objectionable in varying degrees, in proportion to the sensitiveness of the subject and the degree of constriction required. It is true that in many, or in most, cases when the arterial pressure is within normal limits, it is not disconcerting, and is therefore borne with equanimity; but when it exceeds 180 or 200 mm. Hg., in not a few cases it is apt to be resented, and in a case now and then (a sensitive lady, as a rule) the patient will not again submit to the ordeal. In private practice, this is sometimes a real objection to the armlet method, whereas in hospital work it is practically negligible. But discomfort from compression of the arm by the armlet has for us another aspect, and that is its possible influence in fictitiously raising the record of the arterial pressure. We know that pain and discomfort of any kind perturb the whole nervous system, and may raise the arterial pressure by stimulating the ventricle, and by inducing general vaso-constriction. In the majority of cases, any discomfort produced by the armlet is not sufficient to appreciably increase the reading. But can we be sure of this in abnormally sensitive subjects? No one with any experience in manometric observation can doubt that, in many such cases, the readings obtained are fictitious, notwithstanding all the judicious waiting and tact of the observer to reduce to a minimum the nervous perturbation of the patient. Hence the practical importance of minimising the discomfort of the armlet.

Methods of Reducing the Discomfort of the Armlet.—Increased speed in producing armlet compression lessens the discomfort by avoiding congestion in the parts distal to the armlet. This end is secured by adopting one or other of the following procedures:

(1) The observer adjusts the canvas bag (see p. 8) between the filler and the connecting tube, and with the aid of the filler he obtains the initial air pressure of 50 or 60 mm. Hg., and then turns the tap of the filler. He then squeezes the bag, distended as it is with air, until the required degree of pressure is reached. The filling can thus be made as quick as may be desired, consistent with careful observation of the systolic and diastolic pressures. The uninterrupted pressure thus available can be held

with perfect steadiness at any point, and for as long or as short a time as may be wished, and can be let down and raised again under complete control of the hand, and with great facility and exactitude; and the readings thus obtained can be repeated many times for verification or correction, without inflicting needless discomfort from prolonged compression.

(2) The observer fills up to 100 mm. Hg. by the filler, without having the canvas bag in circuit; and then turns the tap of the filler. He now places his right hand over the air-bag of the armlet, and compresses this to just beyond the required reading of the systolic pressure. This quick method is well adapted to observation in sensitive subjects (ladies and others) as well as in all cases in which pressure readings are over 180 to 200 mm. Hg. The observer must, however, be careful to fill up to 100 mm. Hg., and not less, before turning the tap; for it has been observed that a lower initial pressure than this may yield, on hand compression of the armlet, a reading short of that afforded by filling in the ordinary way, or on compressing the canvas bag. This method, though quick and convenient for the reading of the systolic, is not, however, suited to the observation of the diastolic pressure; an objection which may be met by taking the latter

separately as a preliminary or subsequent procedure.[1]

Massage and Recompression of the Brachial Artery by Means of the Armlet.—When the fingers occlude the brachial at the middle of the arm or at the bend of the elbow for some few minutes, the vessel wall is distended by the full impact of the ventricle; and this procedure may suffice in most cases to relax and soften the contracted and thickened artery sufficiently (see p. 129). But, after making the first reading, it is somewhat inconvenient to undo the armlet for the purpose of manipulating the artery. I therefore suggest the use of the armlet itself for the purpose of repeated compression; and this can be done by (1) detaching the rubber tube from the manometer, (2) inserting the plug attached to its end, (3) connecting up the canvas bag (if not already in use), (4) inflating the armlet until the radial pulse rather suddenly swells under the finger, and turning the tap of the filler. The hand now grasps the distended bag and *quickly* raises the pressure just beyond the obliteration point, and then, after the lapse of a minute or two, it releases the pressure suddenly to the initial point. The procedure, which is repeated several times, possesses the following advantages: (1) uniformity of alternate pressure and release over a large portion of the artery, (2) avoidance of irritation from digital manipulation and of discomfort from venous congestion and distension of the limb produced by the ordinary slower method of filling the armlet.

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[1] As a rule it is preferable to make a second decompression reading of the diastolic pressure after letting down the air pressure to zero, so as to allow the congestion of the limb, peripheral to the armlet, to subside and to verify the first reading (G. Spencer Melvin and J. R. Murray, *Quart. Journ. of Med.*, 1914, p. 419).

The Phonendoscope.—The armlet having been adjusted, the thick rubber band (see p. 10) is passed over the forearm to the bend of the elbow, and is adapted to the size required by the metal clip. The observer, having removed the metal shield covering the rubber membrane and placed it over the dome, then slides the phonendoscope under the band over the lower end of the brachial artery, just inside the tendon of the biceps, relaxing, if necessary, the tension of the rubber so as to avoid pressure on the instrument. The rubber band is merely intended to keep the tambour in position during the observation. Finally, the ear plugs are placed *in situ* and the manometer filler and armlet are connected up by the three-way rubber tube.

The Reading of the Pressure.—In all the methods (tactile, oscillatory, and auditory) of reading the arterial pressure the finger should be placed over a distal artery (radial for the arm, and posterior tibial for thigh or calf) to determine the fact of closure by compression, and of reappearance of the pulse on decompression. The oscillatory as well as the auditory method requires to be safeguarded by the tactile criterion of the systolic pressure. Whichever method be followed, the reading should be made by the falling pressure after overstepping the obliteration of the pulse; and not by the rising pressure.

The auditory method with systolic tactile check should be employed in all cases. Should the tactile systolic pressure read higher than the auditory, the discrepancy is generally due to a faulty position of the tambour over the artery; and in that case after readjustment the readings should be in complete agreement, or the auditory one may be a few millimetres higher—as is generally the case. When the throbs are indistinct from excessive deposit of fat or fibrous tissue, they may be rendered more audible by placing the arm on its extensor side and straightening the elbow.

The first reading should not be accepted unless it is corroborated by the second and third. If the readings decrease, they should be repeated until they remain uniform. The quick methods of compression and decompression just described enable us to repeat the observation without causing inconvenience to the patient.

The Reading of the Peripheral Arterial Pressure.—The employment of Gärtner's method on the finger, as an adjunct to the armlet procedure on the brachial artery, is sometimes useful in affording an approximate reading of the amount of the peripheral resistance in the arterial system in hypertension (see p. 58).

The only additional pieces of apparatus required are: (1) a digital air-bag backed with canvas, and (2) a stout rubber ring with a loop which facilitates its removal.

The brachial systolic reading having been made, the digital air-bag is adjusted to the middle phalanx of the middle or ring finger, and the rubber ring is slowly rolled over the finger up to the bag, when the last phalanx will be blanched. The digital bag having been connected with the filler and manometer, the pressure is brought up to the brachial reading just made. Finally the rubber ring is withdrawn from the finger by pulling the loop, when the last phalanx should remain blanched in all cases over twenty (see table, p. 59).

The final stages of the technique are, according to my observations, most important. I think that many of the large variable results obtained by different observers, and by the same observer, are due to a misconception of the long time element which the method requires during decompression, and to the fact that different criteria may be followed, such as either a faint blush or a deep flush or a subjective throb, which afford various readings.

My observations show that four conditions are necessary to secure practically uniform results: (1) Warmth of finger; the preceding brachial observation is, however, useful in removing the vaso-constrictive effect of cold. (2) Decompression in stages of 5 mm. at a time. (3) Very gradual release between the steps. (4) Slowly counting 30, or letting 30 seconds elapse between the steps; the object of this long pause being to secure sufficient time to allow the first diminutive escape of blood to diffuse into and accumulate in the blanched tissue, so that if after the lapse of 30 seconds there should be suspicion that the first escapement has taken place, the halt should be prolonged to a minute to allow of the full development of the blush, which may then acquire the somewhat livid hue of venosity contrasting with the normal rosy tint of the uncompressed fingers.

CHAPTER VII

THE MANOMETER IN OPERATIVE SURGERY, OBSTETRICS, OPHTHALMOLOGY, AND THE SELECTION OF THE BEST LIVES FOR LIFE INSURANCE

I. Operative Procedures

Incisions of the skin and peritoneum produce a transitory rise in arterial pressure from vasoconstriction (Janeway); or a slight fall which is more marked when the anæsthesia is insufficient (G. F. Lull and C. H. Turner [1]). These fluctuations are, however, generally transient and significant, not exceeding 10 mm. or so. But the depression may be serious when nerve trunks or their branches are irritated, severed, or stretched (G. W. Crile,[2] G. F. Lull and C. H. Turner [1]), when the abdominal viscera are roughly manipulated, exposed, or subjected to traction, when an operation is severe or prolonged, or is attended by much hæmorrhage, and when the abdomen or thorax is aspirated for effusion (J. A. Capps and D. Lewis[1]). Manipulation of the pelvic organs causes a rise of pressure proportionate to the severity of the operation; incisions of the *dura mater* do not materially affect the pressure, though irritation of it, by sponging, produces a rapid fall (G. W. Crile[2]). C. G. Wiggins[3] has observed that the manometer is useful in detecting internal hæmorrhage after an operation; its presence and continuance being indicated by a gradual and progressive reduction of the pulse-pressure, combined with a rising pulse rate, and its cessation by a recovery of pulse-pressure and by the lessening of the pulse rate.

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[1] *Therapeutic Gazette,* 1911.

[2] *Blood-Pressure in Surgery,* Philad., 1903.

Shock.—The manometer is becoming a useful guide to the surgeon in detecting the onset of shock, and in following the effects of treatment designed to counteract its progress. In by far the majority of cases of shock a rapid fall of the arterial pressure occurs, and this is the best sign of shock, and a danger signal which cannot be safely ignored. It is difficult to devise a theory of shock which is consistent with all the facts. Three have been suggested by Crile, Yandell Henderson, and Rendle

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[1] *Amer. Journ. Med. Sciences*, 1908.

[2] *Blood-Pressure in Surgery*, Philad., 1903.

[3] *Archiv. Int. Med.*, 1910.

Short; but they all differ in detail. They are, however, in agreement as to the first step in the causation of shock; namely, in the noxious association of pain, peripheral irritation, fear, and anæsthetic poisoning. Crile believes that these depressant causes produce exhaustion of the vasomotor centres in the medulla and cord, which leads to a rapid and pronounced fall of the systolic pressure to 60 or even 40 mm. Hg. in extreme cases, and to retrograde changes in the cells of the nervous system; and he has devised his anoci-association method of preventive treatment which intercepts the up-going stimuli and greatly reduces the impressions of physical pain and the effects of operative manipulation. Since adopting this method, his mortality from shock has fallen from 4·4 to 0·8 per cent. for all operations. The following is a brief summary of the method: (1) Injection of scopolamine gr. $\frac{1}{100}$ and morphine gr. $\frac{1}{6}$ an hour before the operation. (2) Light and even unconsciousness maintained by ether (open method) of which only about a third of the usual amount is required. (3) Infiltration of the skin and of deeper layers by novocaine solution (1 in 400). (4) In abdominal operations, after opening the peritoneum, pack the cut with wet gauze and inject freely the peritoneal edges, which are everted for the purpose, with a solution of quinine hydrochloride, and urea (5 per cent. of each in normal saline).[1]

The prevention of shock by the anoci-association method is of course of greater importance than the treatment of it; but should the latter be required, normal saline transfusion, which maintains the output of the heart,[2] should be resorted to.[3] According to the teaching of J. C. Bloodgood,[4] when the pressure falls to 100 mm. or lower, "it is time to stop the operation and give the saline immediately"; and in view of avoiding post-operative collapse, which not infrequently happens even though the patient seems in fair condition after an operation, a pressure reading should be taken before the patient is transferred to bed, for if this reading is much lower than that recorded before the operation, it indicates that the patient should still be kept on the operating table, and given the salt solution by one or all three methods. Crile regards stimulants, such as alcohol and strychnine, as injurious in shock; and A. Rendle Short[1] supports this teaching, and expresses a doubt as to the efficacy of pituitary extract and adrenaline. The action of all these remedies is either negative or quite transitory.

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[1] *Brit. Med. Journ.*, 1914, vol. ii, p. 113: article by H. B. Butler, F.R.C.S.E., and E. W. Sheaf, M.C., Cantab. For full practical details the reader should consult *Anoci-association*, by C. M. Crile, M.D., and W. E. Lower, M.D., edited by Amy F. Rowland, Philad. and London, 1914.

[2] *Lancet*, 1914, vol. i, p. 732: Hunterian Lecture by Rendle Short, M.D.

[3] The reader may be reminded of the value of transfusion of normal saline when the arterial pressure falls acutely and severely in cholera and in the so-called shock of burns. J. P. Lockhart Mummery regards the latter as toxæmia (*Lancet*, 1914, vol. ii, p. 858).

While some observers rely on intravenous saline solution alone in the treatment of shock, others (Cook and Briggs in *Johns Hopkins Hosp. Reports*, 1903) assert that it fails to raise the arterial pressure unless combined with adrenaline, and some (Pearce and Eisenburg in *Archiv. Int. Med.*, 1910) add a cardiac stimulant—digitoxin—to the adrenaline saline solution (1 adrenaline in 40,000 saline) which is slowly administered.

[4] *Penns. Med. Journ.*, 1912; *Annals of Surgery*, 1912.

The probable value of the transfusion of sodium bicarbonate solution into the veins, in man in a state of shock, is suggested by the recent experimental work of Seelig and his collaborators (J. Tierney and F. Rodenbaugh) on dogs in a state of shock induced by manipulation and chilling of the abdominal viscera; for these experimenters observed a pronounced and well-maintained rise of the arterial pressure, averaging 18 mm. Hg., and not uncommonly 40 mm., following the transfusion, while at the same time the amplitude of the heart's beats and the depth of respiration were increased. Over eleven years ago (1903) Howell[1] found that sodium bicarbonate furnished better and more prolonged pressure results than those obtained from adrenaline, pituitrin, alcohol, and strychnine.

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[1] On the other hand A. Rendle Short asserts that shock has been repeatedly observed to occur after the patient leaves the operating table, with a normal pressure (*op. cit.*).

In the treatment of shock, the application of warmth to the extremities should not be overlooked; and elastic bandages should be applied to the limbs and abdomen—care being taken to avoid embarrassment of the diaphragm—so as to avoid stasis in the veins from deficiency of *vis a tergo* and muscular tone.

The Recording of the Arterial Blood-Pressure and Pulse Rate during Operations.—The charting of the systolic pressure (auditorily read) and the pulse rate should be begun either on the day before the operation or on the day itself, before the preparations for it are made, when the patient is as free as possible from flurry and excitement. During the operation observations should be recorded every five minutes, by a clinical clerk or nurse told off for the purpose. A thick black line should be ruled on the chart at 100 mm. Hg., and the blood-pressure should be entered with a red chalk pencil, and the pulse rate with a black one.

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[1] *Lancet*, 1914, vol. i, p. 335.

The chart should be visible to the surgeon or his assistant, and to the anæsthetist. A final record should be made before the patient is removed from the operating table; and after his removal, further observations should be made every two or three hours, as a precaution against post-operative collapse or internal hæmorrhage. As the risks of an operation are practically unknown in any individual patient, the systematic charting of the arterial blood-pressure and pulse rate should be made in every operation: and, if that is not found possible, it certainly should never be omitted in major operations (especially abdominal) or when it is known that a patient is handicapped by some circulatory weakness or defect. For instance, operations should be undertaken only after grave consideration in people with very high blood-pressure, not only because of excessive hæmorrhage which is likely to occur during the operation, but also for fear of secondary hæmorrhage setting in later.

The Effects of Anæsthetics on the Arterial Pressure.—To follow the variations of pressure produced by an anæsthetic in a normal subject is one thing, and to do the same in the course of an operation is quite another; for in the latter case, the normal effects of the anæsthetic are disturbed by the up-paths of painful stimuli. Manometric observation during an operation is therefore the resultant of two causal factors, the anæsthetic and the operation, and not that of the anæsthetic alone. Much of the conflicting evidence on the clinical effects of anæsthetics on the arterial pressure is ascribable to the varying conditions of operative procedures. Notwithstanding this source of fallacy, in attempting to follow the influence of anæsthetics on the circulation in the course of surgical work, we can, however, recognise the broad lines of that influence, which have been determined by the experimental study upon animals. The conclusions of the majority of anæsthetists indicate that (1) chloroform reduces the pressure, that (2) ether maintains it, after a rise due to mental excitement and struggling, that (3) nitrous oxide raises it from its asphyxial effect, that (4) nitrous oxide combined with oxygen produces a transitory rise followed by the normal level of pressure which is maintained. In England and in America open ether is superseding chloroform; and in America nitrous oxide and oxygen for continuous anæsthesia are displacing ether as much as possible, and may even supersede ether when the special technique is improved, as “ it is the safest general anæsthetic known, being practically non-toxic ” (G. McCall

Smith [1]). The best anæsthetic is that which maintains the pressure at or near the normal level, with the least variation during the operation, and leaves the patient as free as possible from liability to post-operative shock or collapse.

II. Obstetrics

The manometer in obstetrics and pregnancy has been studied with much painstaking thoroughness by J. C. Hirst[2] and other observers who agree with his leading conclusions, which he thus summarises :[3]

(1) The normal blood-pressure in normal, healthy, non-pregnant women will not vary much from 112 mm.

(2) The normal blood-pressure in healthy, pregnant women will average close on 118 mm. A slight increase over this figure is to be expected in the last month of pregnancy.

(3) Blood-pressure in toxæmia, in the first half of pregnancy, associated with pernicious vomiting, is usually low.

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[1] *Lancet*, 1914, vol. i, p. 785.

[2] *New York Med. Journ.*, 1910.

[3] Quoted also in full by F. A. Faught, *Blood-Pressure from the Clinical Standpoint*, 1913.

(4) Blood-pressure in the latter half of pregnancy, associated with albuminuria and eclampsia, is invariably high.

(5) A high and rising blood-pressure is an invariable, and very often the earliest sign of toxæmia, in the latter half of pregnancy.

(6) Upon the rupture of the membranes, there is an immediate fall of pressure of from 60 to 90 mm. This fall is temporary only, but is attended with marked relief in the headache and epigastric pain these patients so frequently complain of. The relief from these symptoms lasts, however, for some hours after the pressure returns to near its original height, which is shortly after the first fall. A similar fall, though much slighter, is noticed after a sweat bath.

(7) There is a second fall from of from 60 to 90 mm. after the child is born. This again is only temporary, and in from fifteen to thirty minutes, if a patient has not bled profusely, the pressure returns to about its level before the birth.

(8) Usually in eclampsia, the pressure remains high for forty-eight hours after the birth, then begins to subside, and reaches the normal of from 118 to 124 mm. in from seven to ten days after delivery.

(9) As far as it is possible to lay down any rules

in these cases, we may say that a blood-pressure of below 124 mm. could be disregarded ; a pressure of from 125 to 150 mm. needs careful watching and moderate eliminative treatment ; and that a pressure of over 150 mm. needs usually active eliminitive treatment, and will, in all probability, especially if it shows a tendency to climb higher, require the induction of premature labour.

Those who have worked on the toxæmia of pregnancy have shown that it produces a rise of pressure of various degrees in the majority of cases, and now and then a lowered pressure ; and that when the rise is moderate, or even undecided, delivery is, as a rule, followed by recovery, but when the pressure is lowered, or is extremely high, the issue is generally quickly fatal.

For treatment and prognosis, periodical manometric observation during pregnancy is most valuable ; and especially in the later stages, with the view of the early detection of toxæmia, when eliminative treatment is of greater avail than at the actual onset of eclamptic attacks, and when the manometer is a valuable guide in deciding on the urgency or otherwise of premature delivery.

" A pressure of 160 mm. during late pregnancy should always be considered highly suspicious and lead to the same careful and repeated observations that the finding of albuminuria does. A further rise, particularly if rapid, and associated with marked headache or vomiting, should probably be the signal for terminating pregnancy. After delivery in normal cases, the blood-pressure falls promptly to normal or below. The persistence of high pressure *post-partum* must always be considered a danger signal, and continued watchfulness should not be relinquished until the blood-pressure is normal. I feel strongly that the bulk of obstetricians have not as yet given sufficient attention to this subject. I believe it will soon be the custom to make blood-pressure observations during pregnancy as frequently as the urine is examined; to make a reading during the stage of labour, of course between the pains; and to make readings subsequent to delivery until the pressure has returned to normal."[1]

III. OPTHALMOLOGY

The principal value of the manometer in ophthalmological practice rests on the frequent causal relation of retinitis and hæmorrhage with chronic interstitial nephritis and arterio-sclerosis; and on the dependence of glaucoma (acute and chronic) on a supernormal arterial pressure.

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[1] *Albany Med. Annals,* 1911: "When should the General Practitioner measure the Blood-Pressure," by Theodore C. Janeway, M.D., M.A., etc.

It is likewise useful in studying variations of the retinal circulation which produce temporary blindness, or mere dimness of vision. When the retinal vessels become spasmodically contracted the onset of the attack is sudden, seizes one retina at a time, and is of short duration—not exceeding an hour; and the arterial pressure is markedly supernormal. But when the vessels are atonic, the atony may be associated with lowered pressure—especially diastolic.

A sub-conjunctival hæmorrhage, especially in the elderly, is not infrequently a tell-tale of a supernormal arterial pressure.

The detection of a high pressure before operating on the eye, *e.g.* for glaucoma, and for extraction of a senile cataract, is important; for unless modified by rest and other treatment, the increased pressure may induce intra-ocular hæmorrhage after the operation.

IV. Selection of Best Lives for Insurance and Public Services

The large and increasing mortality from vascular diseases, on which efficiency of life-work and longevity so largely depend, emphasises the importance of increased vigilance and thoroughness in the examination of lives for insurance; and also for various public duties, in which the capacity of life possesses a certain social and financial value.

In life insurance this question is of great importance, not only for the companies, but for the insured; for, the sounder the accepted lives the lower should the rates become.

It is somewhat remarkable that for some years past many of the large life-insurance companies in the United States have appreciated the practical value of the manometer in the examination of lives for insurance, and have directed their examiners to employ it in every case; whereas in this country the instrument has not been adopted, so far as I know, by any insurance company as a necessary requisite for the examination of prospective lives, though doubtless many of their examiners on their own initiative do use the manometer as an additional aid in estimating the integrity or the impairment of the cardio-vascular system.

Now that some years have elapsed since the American companies adopted this innovation, facts have accumulated which may ultimately lead our companies, in their own interests and in those of the insured, to revise their position in regard to this matter.

CHAPTER VIII

THE TREATMENT OF SUPERNORMAL ARTERIAL PRESSURE

In attempting to control or modify a rise in the arterial pressure we should keep in mind that, though variations of that pressure may produce certain pathological effects, they themselves are but results. When, for example, the arterial pressure is raised but *temporarily*, the cause may be, and, in fact, usually is, of nervous origin—either psychical or reflected; but when the pressure rise is *persistent* it may be regarded as the outcome generally of some alteration in the chemical composition of the plasma or of organic changes in the circulatory mechanism, which may or may not have themselves resulted from some biochemical cause. It follows, therefore, that in our efforts to control a persistent rise in the arterial pressure we should, if possible, learn what are the etiological factors at work. Unfortunately, as our knowledge in this direction is extremely limited, we must often content ourselves with symptomatic treatment, or treatment on general lines, with the application of such practical suggestions as may be derived from physiology, which is, after all, the mother of medicine.

I. The Psychical Management of the Patient

We are sometimes apt to forget that when a patient of a nervous, sensitive type realises that his arterial pressure is raised, he is apt to exaggerate the import of this knowledge, and may worry—often quite unnecessarily worry—himself about it. I have met with several such cases. We all know that our nervous patients are apt to nurse their pet worries ; but when the arterial pressure is raised, perturbation is more particularly injurious, for it tends to perpetuate the evil. We should therefore do our best to dispel apprehension, and to instil into our patient as much hope and encouragement as may be possible.

It is a good rule, when treating nervous subjects, to abstain from quoting the actual figures of an observation, especially when they are high (*e.g.* 200 mm. Hg. or more).

II. DIETARY TREATMENT

Reduction of Gastric Cardio-vascular Stimulation.—The principal aim in our dietetic directions should be to select a diet which has the least stimulating effect on the circulatory organs. Hence we should reduce as much as possible not only the bulk of each meal, but also the proportion of those constituents of the foodstuffs which more particularly stimulate the cardio-vascular apparatus, such as various salts, soluble extractives, active principles and other bodies (*e.g.* alcohol), which are directly absorbed without change, and may be regarded as exogenous hormones between the digestive tract and the circulatory apparatus, stimulating the heart on the one hand and the capillary circulation on the other.

In the normal state of the circulation the vasomotor play of the peripheral parts is free, and responds to the variations in the heart's action; but this harmony becomes disturbed in cases of supernormal arterial pressure, in which the stress of any stimulation of the circulation falls more and more on the heart muscle, with increment of the arterial pressure. Hence we should in such cases try to modify as much as possible the cardio-vascular stimulation produced by the ingestion of food.[1]

The next important point is to correct digestive irregularities [and examine the mouth for carious teeth and pyorrhœa, which commonly cause gastric disturbances and possibly by the absorption of toxins contribute to a rise in blood pressure.—A.M.]. We should encourage extra mastication and insalivation, especially of the amylaceous foodstuffs, which are generally so soft as not to seem to need any mastication at all, and are consequently bolted, whereas it is more important thoroughly to masticate and insalivate them than the protein foods.

The supply of meats of all kinds should be lessened below the proportion ordinarily taken, and some are satisfied merely to reduce or to disallow the red meats (beef and mutton), while they permit the taking of white and young meats (fish, poultry, veal, etc.) in ordinary quantities. Though the practitioner nearly always is inclined to select what are called lighter meats, I cannot say that I have observed any difference of effect on the arterial pressure of the various forms of roast or fried meats—whether as beef, mutton, fish, poultry, etc. A more important difference in this respect seems to me connected with the way in which the meats are cooked—whether roasted and taken with the gravy, or boiled and served without the broth. I therefore make it a rule in these cases to substitute as much as possible boiled for the roasted or fried forms of fish, fowl, beef, mutton, etc.; and to exclude from the dietary meat extractives, such as meat soups, gravies, etc. The proportion of green vegetables and fruits should be increased, and carbohydrate foods may be taken in moderation. As a rule I think the cases do best on the reduction of an ordinary diet as a whole, with a diminution of the animal and an increase of the vegetable foodstuffs. The reduction of the ingesta to physiological needs and expenditure is particularly important in the management of cases of high arterial pressure; for some of these patients are inactive and, feeling weak and run down, crave for the stimulant effects of tonics, a full meat diet, an excess of salt and other condiments, and alcoholic drinks.

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[1] See some good illustrations of the value of this teaching in a paper by William Russell, M.D., *Brit. Med. Journ.*, vol. i, 1906.

The Ingestion of Fluids—their nature and quantity—is not a matter of indifference. The bulk of the meals should be kept down by reducing the quantity of fluid taken with them. Fluid, of which the best is water, should be taken more freely when the stomach is empty—*e.g.* a tumbler an hour before meals, or night and morning. Preference should always be given to soft or distilled and non-aerated water, or to mildly alkaline waters, as potash and soda salts improve the peripheral circulation; and it is best generally to advise the water to be taken warm. Hard water—*i.e.* water containing calcium salts in such proportion as to raise the hardness above five degrees—is best avoided, especially in the elderly with a premature rise in the arterial pressure, in whom there may be an inadequate elimination of calcium salts by the bowels and kidneys. Though calcification is generally believed to follow and not to precede degeneration or involution, it may turn out that inadequate elimination may lead to the deposition of calcium salts in the arteries, kidneys, and other tissues of the aged.[1] As the combination of calcium with an organic acid (*e.g.* citric) deprives the calcium ion of its specific physiological effect on the blood,[2] the addition of lemon juice to hard water may be a useful corrective in the elderly and gouty.

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[1] An interesting communication, "The Part played by the Calcium Salts in the Blood and Tissues," appears in the *British Medical Journal* of April 20, 1907, in which W. Blair Bell, M.D., refers to three calcium physiological periods. "The third period is that of late life, when no calcium salts are required for the building up processes or for those of reproduction. These salts then accumulate in the tissues, especially in the vessels . . . atrophy occurs, and the individual gradually declines."

[2] See J. Coleman, *Bio-Chemical Journal*, April 1907.

The beverages, such as tea and coffee, are thought by some observers to be particularly noxious. Long-continued indulgence in strong tea and coffee is undoubtedly injurious, but I have seen no objection to these beverages in strict moderation, and when their ingestion is not followed by disturbing results, such as palpitation, flushing, giddiness, or wakefulness. Cocoa is the least objectionable of beverages. As to alcoholic beverages my impression is that high-pressure cases as a class are better without them, but that many cases in which we have moderate or slight increments of pressure may be allowed small quantities without injurious effects, especially if, as a rule, we exclude wines and malt liquors. There is no doubt that when the vaso-motor system is intact, alcohol (as in the form of whisky or gin) is vaso-dilating and has but a transitory effect in raising the systolic pressure. Experimental work on alcohol by W. E. Dixon of Cambridge[1] shows that in moderate doses alcohol in normal animals and also in man may raise the systolic pressure a little (and especially in animals showing signs of circulatory failure), though it may not do so, while it always diminishes the diastolic pressure (" in other words the difference between the systolic and diastolic pressure tends to increase "), and that it increases the activity and output of the heart, an effect which is ascribed to the fact that alcohol is a readily assimilable food substance.

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[1] "The Action of Alcohol on the Circulation," by W. E. Dixon, M.A., M.D. Lond., *Journal of Physiology*, March 1907.

Chloride of sodium as a constituent of foodstuffs and as a condiment deserves some notice in the treatment of supernormal arterial pressure. The work of Widal and Javal[1] and other Continental observers has shown that superchloridation and superhydration of the tissues take place when the kidneys become impaired in their permeability to the passage of chlorides, as in chronic nephritis and probably in other phases of renal inadequacy not recognised as organic; and that some of the manifestations of Bright's disease and heart disease (*e.g.* œdema, albuminuria, etc.) are closely associated with this tissue retention of chlorides and water and are relieved by a dechloriding diet. At present I cannot say from my own observation what is the rôle, if any, which the retention of chlorides plays in the production of the high ranges of arterial pressure which we meet with in chronic interstitial nephritis. Probably it is insignificant; but I think it advisable to test the matter by observation. To apply efficiently a dechloriding diet the mere omission of salt as a condiment is insufficient. We must besides this select those articles of food which in the uncooked state contain the least percentage of salt, and omit the addition of salt in their preparation for the table. The diet thus obtained is practically, though not absolutely, chloride-free. Such a saltless diet may consist of vegetable soups (made without meat stock), fresh green vegetables, fruits, nuts, fats (fresh butter, cream), salt-free bread, farinaceous vegetables (potatoes, rice, peas), and sugar. Dufour (in 1905), Guinon and Pater (in 1906), and Déléarde (1907) obtained excellent results from a dietary like this in scarlet fever and scarlatinal nephritis. But in chronic nephritis and in other cases of high arterial pressure such a strict chloride-free diet will scarcely be necessary, though it may perhaps be adopted with advantage for a while now and then, when accessions of pressure arise. In fact, a saltless diet soon becomes so distasteful to the majority of patients that it is difficult for them to continue it for more than a week or two at a time.

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[1] *La Cure de Déchlorisation*, par le Dr. Fernand Widal et le Dr. Adolphe Javal, Paris, 1906.

I have observed that as we grow older (say over sixty-five years of age or so) there is apt to arise a tendency to indulge more and more in the use of salt. When the arterial pressure rises unduly in elderly subjects we should do well to look out for this disposition. The suggestion of the late Sir W. Roberts, that we should substitute chloride of potassium for chloride of sodium as a table salt, is supported by Bunge,[1] who has shown that when potassium is ingested in excess of sodium a loss of the latter from the system takes place. Unfortunately, chloride of potassium is by many disliked as a table salt, but it is less objectionable when combined with 50 per cent. of sodium chloride, or with potassium nitrate. The following formula of an apressor table salt may be found useful: ammonium hippurate, 1 part; potassium nitrate, 4 parts; potassium chloride, 25 parts.[2]

A strict milk diet, sufficient in quantity to maintain the health and condition of the patient—*e.g.* six pints a day—supplies to the system from 100 to 200 grains of salt (different authorities giving the salt percentage of milk as ranging from 1·6 to 3·0), so that such a diet is by no means saltless. Nevertheless, experience shows that a milk diet or a lacto-farinaceous diet for a few weeks has sometimes an excellent effect on high arterial pressure and on the general health.[1] Unfortunately, milk is often badly borne in some cases, especially when the bowels are inclined to constipation and the liver is inactive; but even then it may often be made to agree by adding to it citrate of soda or potash, or when there is gastric acidity sodii bicarbonas or liquor magnesii carbonatis. But a lacto-vegetarian diet is more suitable in most cases ; a moderate daily quantity of milk (*e.g.* two or three pints) when taken with vegetable food-stuffs (bread, rice, green vegetables, and fruits) should not seriously raise the low percentage of ingested salt and, moreover, it advantageously increases the proportions of the protein and fat, especially when combined also with cream, cheese, and eggs. Cheese in moderation is perhaps the least objectionable form of protein food in these cases. Meats of all kinds contain even a smaller percentage of native chloride than milk ; but the cardio-vascular stimulant effect of the extractives which they contain more than counterbalances any advantage they possess in their relatively low percentage of salt. Boiled meats, however, containing less salt and extractives than roast meat, may be added to the dietary without disturbing its comparatively salt-free character. Fresh-water fish (such as trout, trench, pike, and crayfish), which contain much less salt than salt-water fish, may also be taken if we do not require to adhere to a strict minimum supply of ingested salt. Caviare, pickled salt-water fish, molluscs, and shell-fish should be avoided. The condiments—pepper, mustard, vinegar, lemon, etc. —when taken in moderation, are useful in relieving the insipidity of the dechloriding diet.

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[1] G. Bunge, *Text-book of Physiology and Pathological Chemistry* (translated by F. A. Starling), 1902.

[2] Messrs. Oppenheimer, Son & Co. prepare a portable form of this salt.

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[1] See an address on Arterio-sclerosis by Sir W. P. Herringham, M.D., *Brit. Med. Journ.*, vol. i, 1907, p. 61. According to the recent experiments of C. J. Coleman, milk as a diet does not appear to increase the coagulability of the blood, as much of its calcium content is not available as a factor in controlling the coagulation, for it is probably combined with *organic* (and not *mineral*) acids, which combination does not shorten the coagulation time (*The Bio-Chemical Journal, op. cit.*). So it is not improbable that if calcium favours a rise of arterial pressure (*vide seq.*) milk may fail to do so, just as it fails to increase the coagulabilty of the blood.

When the systolic arterial pressure maintains a high range this dietary should be tried, and especially when there are albuminuria and any œdema, however slight. But even when the arterial pressure is equally high and there is not even the merest trace of albumin in a pellucid urine, we may still entertain a suspicion of kidney insufficiency—such as may diminish the permeability of the chlorides ; and we may find a trial of the dechloriding diet as useful in such cases as in the albuminuric ones. When the kidneys are sound and normally permeable to sodium chloride it is not likely that the ordinary use of salt will interfere with the eliminatory work of the kidneys or will raise the arterial pressure, except quite temporarily; for normal renal elimination of the chloride is too rapid to allow of a permanent rise in the percentage of it to take place in the plasma and tissue fluids. We may therefore infer that if there is a permanent retention of chlorides it will be invariably pathological and not a physiological condition, and our aim in treatment is to restore the chloride equilibrium by reducing the amount of the ingested chloride to that which is eliminated by the kidneys. The question, therefore, is one of renal inadequacy in the elimination of the chlorides.

Should the arterial pressure be permanently raised without there being any failure in the elimination of chlorides, urea, and purine compounds, the reduction of ingested salt is not likely to lower the arterial pressure—though it is well to test the matter by observation.

Tobacco smoking is sometimes looked on as a questionable habit when the arterial pressure is increased, and knowing that nicotine raises that pressure and will even produce something like arterio-sclerosis in animals we may be disposed to look on smoking with suspicion. But smoking does not necessarily imply the absorption of all the nicotine contained in tobacco—as when, for example, the weed is chewed. For combustion breaks up the nicotine into such products as pyridine, carbon monoxide,[1] etc., which predominate in the smoke, though this may also contain a little nicotine. Hence the physiological effects of smoking on the arterial pressure will be due to the combined action of all the products of combustion as well as those portions of nicotine which escape destruction and which may be directly absorbed from the end of a cigarette or cigar. From the observations which I have made on myself and other normal subjects not habituated to smoking, I find that it always raises the systolic pressure (*e.g.* from 10 to 20 mm. Hg.) without producing a corresponding rise in the diastolic pressure; in fact, the latter pressure will generally either remain unaltered or may even fall, the pulse pressure range (*i.e.* the difference between the minimum and maximum pressures) being increased. The effect, as a rule, passes off quite quickly, generally in ten minutes or so; but notwithstanding this fact, it is undesirable in the management of cases of supernormal arterial pressure to permit indulgence in a habit which raises the systolic arterial pressure. Besides, Barazzoni found that in subjects with high-tension pulses the rise of the tension produced by tobacco smoking is more exaggerated than in subjects with low or medium tension, and leads to dyspnœa, palpitation, præcordial distress, vertigo, tremors,[1] etc. In most cases, when the habit of smoking can be given up, it is best to encourage the patient to do so ; and abstinence can generally be easily enforced in those who have been light or occasional smokers or who experience slight palpitation, heart hurry, anginal pain, or other discomfort after smoking. But in the habitual smoker it is not always easy even to limit the indulgence, though we should endeavour to do this whenever possible.

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[1] See *Lancet*, Jan. 2, 1904, p. 43; and Feb. 6, 1904, p. 395 (J. S. Thompson, M.D.).

[In excessive cigarette smoking accompanied with inhalation I have not infrequently found a low blood-pressure, but with a considerable reduction in the number of cigarettes used or after actual total cessation a gradual rise has been observed in the pressure.—A. M.]

III. Rest and Exercise

The value of absolute rest in the management of high-pressure cases should be emphasised ; for

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[1] *Gazzetta Medica Italiana,* 1905.

I doubt if we all quite realise it as fully as we ought to do. We not infrequently see its calmative influence on the arterial pressure of bed cases (as in hospital), the pressure readings in similar pathological conditions being generally lower in them than in patients leading their ordinary lives. Absolute rest is the best sedative of exalted ventricular action, that large factor in maintaining supernormal pressure, especially in the higher ranges; and this is often apparent even when the response of the peripheral arteries to a vaso-dilator is either greatly limited or is not apparent. In many such cases increments of pressure recur from cardiac excitation induced reflexly or biochemically, when recumbency for varying periods is invaluable, even though the heart muscle may not show signs of actual failure. The periods of absolute rest required may vary from a day once or twice a week to even three or four weeks at a time[1]; and after the lapse of the longer interval of resting care should be taken to see that the asumption of the accustomed exercise is gradual, or the good effects of the recumbency may be nullified.

Then, on the other hand, the prescription of exercise is important.[1] When the arterial pressure rises beyond a certain moderate degree the patient is apt to become somewhat lethargic and inactive. We should in such cases encourage the taking of as much regular, moderate, and equable exercise as the patient's limit will safely permit. Walking is the best of all exercises, but, unfortunately, even when indulged in quite moderately, these patients frequently complain of early "fagging." Then in suitable cases gentle cycling on roads of low gradients and with a low-geared cycle is preferable, or horse riding—a form of exercise which we are apt to overlook in this mechanical age. All sudden forms of exertion should be avoided, such as straining at stool, etc. But the prescription of exercise when there is a rising pressure in the elderly requires discrimination and caution. Many men over sixty years of age, who have led sedentary lives, on retiring from business are apt to adopt the ways and games of young men, and, being unconscious of the fact that they have already used up much of their endowment of rubber in their arteries, are apt to come to grief in cardiovascular breakdown. Such cases are an object-lesson to us to preach moderation in physical exercise to the elderly in whom we detect signs of diminished elasticity and play in the vascular walls, so that they may be induced to economise their remnant of rubber. Still, on the other hand, moderate activity in the elderly is generally beneficial, and we should always remember that after middle life involution and degeneration are fostered by inactivity; hence a sudden retirement from active business often leads to a premature break-up. The cardio-vascular stimulation of exercise should always be avoided just after a meal. The patient should then rest recumbent or semi-recumbent for a good part of an hour; and after that interval has elapsed he should always take exercise, however limited it may be. Special exercises, such as tension exercise, and graduated resistance and slow deep breathing exercises, are often valuable in widening the peripheral vessels and in letting down the arterial pressure; and massage is useful when the heart begins to weaken and is not equal to efficient active exercise.

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[1] During the prescribed rest evacuant treatment of the liver and bowels should be more particularly maintained.

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[1] We have seen that rest may lower the arterial pressure by calming an overtaxed ventricle in a state of irritable activity. On the other hand, the moderate exercise of the muscles may also diminish the pressure by: (1) oxidising and destroying the adrenaline which maintains their tone and, by accumulation in the blood, raises the arterial pressure; (2) generating lactic acid and perhaps other depressor metabolic products; and (3) dilating the peripheral circulation.

IV. BALNEOLOGICAL TREATMENT

Some experience in the use of different kinds of baths in the treatment of these cases has clearly indicated to me that some balneological procedures are more potent than others in relieving supernormal arterial pressure. Immersion in warm fresh water lowers the arterial pressure chiefly by relaxing the cutaneous and splanchnic arterioles ; but this effect is quite transitory, and the cutaneous circulation is left more amenable than before the immersion to the contracting influence of a lower temperature. But when the water contains or is charged with certain chemicals (such as chlorides, sulphides, extract of pine, peat, and carbonic acid) the lowering effect on the arterial pressure is prolonged. Hence the calmative influence of peat, sulphur, pine, saline, and carbonic acid baths, followed by dry, warm packing on the circulatory organs and the nervous system, and their adaptability to the treatment of those cases of increased arterial pressure associated with some phase of nervous irritability, or with contraction of the cutaneous blood-supply and some inefficiency of the skin as an emunctory organ. A course of such bathing will produce good results, even when the arterial pressure is moderately increased. But

I rarely find it desirable to rely upon these still baths entirely, for they are apt to leave the muscular system relaxed and toneless. They are more valuable as intercurrent baths in a course of massage bathing, which exerts a more decided effect in lowering the arterial pressure while maintaining or even improving the nutrition and tone of the muscles. Warm massage douching, exemplified by the Aix-les-Bains douche, followed by the needle bath of alternating temperature and warm pack, is, according to my observation, one of our most valuable balneological procedures for treating cases of supernormal arterial pressure. In it we have in synergic combination the action of warmth, massage, and vibration in diverting the blood to the peripheral vessels of the richly endowed vascular area of the muscles as well as that of the skin.

V. Electrical Treatment

My experience of the therapeutic value of electricity in cases of supernormal arterial pressure is somewhat variable. It has scarcely given me sufficient confidence in its efficacy to encourage me to rely on it as the sole mode of treatment beyond the dietetic and general management.

But I have not infrequently found it to be a useful adjuvant when resorted to along with other balneological procedures.

Radiant electric light baths, which secure active cutaneous elimination at a comparatively low temperature, have proved most useful in many of my cases; and I frequently advise their continued use twice or three times a week.

The D'Arsonval current for twenty minutes daily, with or without bipolar massage, or the electric breeze, or the sparking application to the limbs and body, has occasionally afforded me encouraging results. Montier has adduced some rather striking evidence in support of the reducing effect of D'Arsonvalisation in hypertension; he having, for example, observed in some cases, even after a few *séances* only, a fall from such high readings as 220 or 230 mm. Hg. to 150 or 160—a fall which was, moreover, remarkably persistent notwithstanding the discontinuance of further electric treatment.[1] I have not myself observed such satisfactory results.

Schnee's four-cell electric bath (galvanic or sinusoidal) is another form of electrical treatment which I have found useful in hypertension, especially in chronic gouty subjects.

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[1] *Journal de Physiothérapie*, 15 *août*, 1905.

VI. Care of the Skin

It is important in these cases to maintain the cutaneous circulation at its optimum efficiency. Warmth diverts to the skin a comparatively large volume of blood, which lowers the general arterial pressure ; hence the value of warm efficient woollen clothing and of warm equable climates (such as those of Jamaica, India, or Egypt) during our winter and spring months. Cold, and especially cold windy climates, are most injurious in high-pressure cases.[1]

Cutaneous elimination should be encouraged ; a thorough perspiration should be obtained once or twice a week by means of a lamp bath (home Turkish bath), or an incandescent light bath, or a Turkish bath, followed by efficient massage and removal of effete epithelium. By active perspiration the plasma and tissue-fluids are relieved of an excess of sodium chloride and metabolic residua.

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[1] High altitudes, even though the air is still, are contraindicated in these cases ; chiefly because of the strain thrown on the heart by the cold and the accelerated breathing necessitated by the altitude. Hence the injurious effects are more apparent when the heart muscle is beginning to fall and when the breathing is easily disturbed by exertion. Sheltered moderate altitudes are, however, sometimes beneficial in hypertonia and supernormal pressure when the heart is fairly equal to its work.

VII. Venesection

So far I have had no experience of venesection in the ordinary course of treatment of high-pressure cases, apart from the actual accidents of uræmia and cerebral hæmorrhage. Now that we have in the clinical manometer an instrument of precision to guide us in selecting the right case for venesection, should we not avail ourselves of the rapid hypotensive effect of this measure more often than we do when suspiciously threatening symptoms arise, or when in certain plethoric subjects a very high pressure does not respond sufficiently to the ordinary preventive measures, especially if the free use of vaso-dilators is not effectual? Under such conditions many years ago I used to apply Junod's boot, with apparently favourable results; but I think a limited venesection would be still more effectual than this. The apparatus acts like a magnified cupping-glass; it consists of (1) a metal boot sufficiently large to accommodate the leg up to the thigh, where the opening is rendered air-tight by a broad band of indiarubber, and (2) an air-pump attached to a nozzle communicating with the interior of the boot through which the air within is rarefied.

VIII. MEDICINAL TREATMENT

Sometimes dietetic treatment, regulation of rest and exercise, and some balneological procedure—such as thermo-massage—will suffice to correct the increment of arterial pressure, more especially in its slighter degrees. But in most cases it will be found that some medicinal treatment or other in addition will be either useful in expediting the progress or will be necessary. That treatment may be directed into one or other of the following lines—namely (1) to secure (*a*) free evacuation and correction of the liver and bowels, (*b*) gastro-intestinal antisepsis, and (*c*) sedation of the gastro-cardiac reflex; and (2) vaso-dilatation and sedation of the vaso-motor nervous system.

Evacuant and Antiseptic Treatment.—There is no doubt that nearly all forms of supernormal arterial pressure are benefited less or more by purgatives; not so much, however, by the occasional aperient dose as by a daily increased discharge from the bowels. The benefit thus derived may be ascribed to (1) the stimulation and correction of the liver and other abdominal glands; (2) the daily baling out through the intestinal mucous membrane of salt[1] and water from the blood and the tissues, with stimulation of the intermediary circulation; and (3) the removal of intestinal toxins. To be efficient the aperient must produce a free watery discharge. Saline purgatives should be taken in hot water in the early morning, such as sodii sulphas, or soda tartarata, or sodii sulphas with magnesii sulphas; and, in addition, there should be an occasional mercurial dose—*e.g.* once or twice a week (calomel or pilula hydrargyri) at bedtime. Just now there is a widely entertained belief that the generation of toxins in the gastro-intestinal area (chronic intestinal auto-intoxication) is the possible, if not probable, cause of several ailments of obscure origin, such as gout, arterio-sclerosis,[1] etc. Metchnikoff has shown us the importance of avoiding the

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[1] The normal proportion of salt in the fæces is greatly increased in

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diarrhœa. *Vide* "L'Elimination du Chlorure de Sodium par la Diarrhée" (*Comptes Rendus de la Société de Biologie*, 1903, p. 929).

[1] A few years ago I noted the arterial pressure of patients suffering from colitis, who have undergone the Plombières treatment at Harrogate. In those in which the pressure was raised I cannot say that I have observed any marked or appreciable fall of it which could be ascribed to the improvement of the colon, even in the most successful cases. Moreover, I have met with several cases of pronounced chronic colitis affording a somewhat low arterial pressure, or certainly without any rise whatever over the normal pressure. From the study of these cases I cannot say that I have discovered any direct evidence showing that colitis is pathologically connected with arterio-sclerosis, gout, or a supernormal arterial pressure. I am therefore rather disposed to doubt if the colon is responsible for the production of a pressor toxin,

ingestion of bacteria in raw fruit and vegetables by heating and cooking them. But cooking is objectionable in depriving certain raw fruits and vegetables of their delicate and appetising flavour. In the case of grapes, salads, and perhaps strawberries, we may, however, render them aseptic without impairing their natural flavour by dissolving one of Dr. Rideal's tablets of bisulphate of soda in ten ounces of water and using this as a wash water. Intestinal antiseptics may often be used with advantage when the arterial pressure is persistently raised, such as benzo-naphthol, salol, salacetol, and tylmarin (acetylated ortho-coumaric acid), which tend to ease the peripheral circulation like the other members of the aromatic series. Mercurials are also not only antiseptic, but relax the arterial wall, and I have observed that parvule doses of calomel (*e.g.* tabloids of $\frac{1}{10}$th grain) twice or three times a day may be taken for a few weeks now and then with a favourable effect on the arterial pressure. The red oxide of mercury tabloids ($\frac{1}{20}$th grain) are also worthy of a trial. Small daily doses of sulphur —such as the compound sulphur or the guaiacum and sulphur tabloids—are not only useful as laxatives but also favour a fall of the arterial pressure.

2. **Sedation of the Gastro-cardiac Reflex.**—In many cases of supernormal pressure of the parox-

ysmal type, as in angina pectoris, it is of some importance in the medicinal treatment to soothe the nerve-endings in the stomach. This may be attained from the medicinal standpoint by administering before meals, when the stomach is empty, such remedies as bismuth (carbonate, trinitrate, salicylate), cærium, hydrocyanic acid, sodium bicarbonate, magnesium carbonate, small doses of liq. morphiæ [or preferably bromide of sodium and glycerine of carbolic acid—A. M.], in those combinations which seem to the prescriber to be best adapted to the case in hand ; and it is often advisable to continue such treatment for lengthened periods.

3. **Apressor Remedies.**—Arterial pressure may be reduced either by diminishing the output of the ventricle or by widening the bed of the whole arterial system, and especially its peripheral portion. Experience shows that it is futile, if not injurious, to attempt to obtain a depressor effect and to maintain it by drugs, which lower the action of the heart. Coley, of Philadelphia, for example, has shown that such drugs as aconite, veratrine, antimony, and chloral are unsatisfactory and scarcely justifiable remedies for the purpose of reducing the arterial pressure.[1] Our choice of apressor remedies is therefore restricted to those which favour dilata-

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[1] *Brit. Med. Journ.*, 1906, vol. ii, p. 1454.

tion of the calibre of the arteries, and thus ease the flow of blood through the distal portion of the circulation. Their physiological action on the arterial pressure (distal and proximal) is easily demonstrated by the manometer in man by administering a dose of nitro-glycerine ; and I have elsewhere shown how useful it is in the course of clinical observation thus to gauge the vaso-dilator response of the arteries.

Desideratum in an Apressor Remedy.—Control of a persistently high arterial pressure by drugs is more or less unsatisfactory. Should an impression be made, there is the gradual failure to maintain it, or the great liability—often, indeed, the certainty—of the recurrence of the higher pressure on discontinuing the remedy or on reducing the dose of it. The effect is, indeed, rarely curative. The desideratum in an apressor remedy seems to be the discovery of some agent which will modify arterial pressure on the lines followed by nature—some product or other which may be continued without harm so long as it may be required. We may, as experimental work proceeds, find the solution of this question in physiological chemistry, and we are already beginning to realise that arterial pressure may be the resultant of the interaction of pressor and apressor products supplied to the blood by the internal secretions of the various organs and tissues, and that the higher ranges of pressure we meet with may arise from the predominance of the pressor products, the anti-bodies being generated in insufficient quantity.[1]

The Depressor Effect of Ingested Thyroid Gland.—We know that the adrenal and the pituitary glands yield pressor bodies; but our knowledge of apressor secretory products is much more limited. Extracts of most organs cause on intravenous injection a transitory fall of arterial pressure; among these the thyroid calls for special mention, for I have observed that the ingestion of the thyroid gland increases the calibre of the arteries and lowers the arterial pressure, and in many cases of increased arterial pressure it is sometimes useful, especially in subjects over forty-five or fifty years of age of a gouty or obese type; but it is not a remedy which should be prescribed in a routine way in the majority of cases. When given in guarded doses with each meal, it will often modify the pressure and improve the general health, and then it may with occasional supervision be safely continued with advantage for long periods. On the other hand, I have seen quite small doses of thyroid produce cardiac irregularity and dilatation in arterio-sclerosis. The supervention of precordial uneasiness, palpitation, or irregularity of the heart's action should at once suggest the discontinuance of the remedy or the reduction of the dose. Thyroid is sometimes better borne when combined with a small dose of strophanthus or digitalis; and it may be advantageously associated with a little free iodine (tinct. iod.). It has been thought by some (Stockman, Rolleston[1]) that the iodides exert their beneficial effect on arterio-sclerosis and heightened arterial pressure by stimulating the thyroid; they do not, however, reduce the arterial pressure. Chalmers Watson[2] suggests that the good results obtained from a bread-and-milk diet observed by Herringham[3] in certain cases of arterio-sclerosis with increments of arterial pressure arise from the profound influence of a diet, in which milk largely preponderates, on the structure of the thyroid gland which he has demonstrated in animals—this therapeutic effect being, according to him, "indirect, the thyroid gland spontaneously recovering under the influence of lessened strain on its functional activity."

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[1] The accumulation of pressor products in the blood may also depend on insufficient destruction of them, *e.g.* adrenaline by the liver.

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[1] *Clinical Journal*, 1905.

[2] *Brit. Med. Journ.*, vol. i, 1907.

[3] *Op. cit.*

The Depressor Effect of some other Normal Constituents of the Plasma.—But I think we may question whether the internal secretion of the thyroid is the only apressor agent present in the plasma, notwithstanding its obvious importance. Is it not probable that the arterial pressure is normally modified by some of the saline constituents, some tending to raise the pressure, while others lower it in proportion to their preponderance? The quantitative relation between the various salts of the serum is maintained within certain limits in health; and in disease that relation may at times be disturbed either by the undue ingestion or the defective elimination of certain salts (such as calcium carbonate and sodium chloride). Somewhat cognate physiological facts point to the important influence of the salts of the plasma. Ringer has shown that the salts of calcium, sodium, and potassium must exist in an ideal circulating fluid in the right proportion, and those of calcium are of singular importance in maintaining the contractility of the ventricle; and Waller has demonstrated that potassium depresses and calcium augments the variations of electric response in nerve. Moreover, we also know the important rôle played by calcium in the coagulation of blood and milk. May not a similar relationship subsist between the various salts of the plasma and the blood pressure ? In man the depressor effects of the potassium salts, whether as carbonate or citrate, on the arterial pressure are often more striking than might be expected from the results of experimental work ; and the maintenance of a lower level of pressure thus obtained may be partly due to the corrective effect of the ingested alkaline salts on the disturbed balance produced by the predominance of pressor salts. In practice we frequently combine the nitrates and nitrites with the carbonates or citrates of potassium. Sir Lauder Brunton associates nitrate of potash (20 grains) and nitrate of sodium (from a half to two grains) with this alkaline treatment (potassium bicarbonate) in a large tumbler of water in the early morning ; and I have observed excellent results from a continuance of this prescription in many cases of decided super-normal pressure. Alkalies are generally well borne by such cases for long periods. But my observations in man dispose me to conclude that there may be other salts normally present in the plasma which tend to lower the arterial pressure, such as the hippurates and benzoates. I gave these salts a good clinical trial some years ago, and I was satisfied that they are useful in many cases of hypertension and of moderate increments of arterial pressure. As a rule they improve the general health and well-being, and are well adapted for long-continued use in the preventive treatment in the hypertonia of goutiness and arteriosclerosis and especially in the elderly. In many cases of hypertension, especially in women and overworked anxious men, I have observed that the association of ammonium bromide with ammonium hippurate or benzoate is still more beneficial in providing a higher degree of sedation of the vaso-motor nervous system than is furnished by either remedy alone. There are also other remedies furnishing hippuric acid to the system which are worthy of trial in cases of supernormal arterial pressure, such as quinic acid, styracol, tylmarin, which are obtainable in the tablet form. It is interesting to find how many remedies with a benzene nucleus are useful in gout as well as in arterial hypertension.

Exogenous Apressor Remedies.—The most important of these vaso-dilator remedies are the nitrites of amyl, isobutyl, glyceryl, sodium, erythrol, and mannitose; all of which vary in the energy and duration of their vaso-dilator activity. The most active members of the group, amyl and isobutyl nitrites and nitro-glycerine, have the shortest dilating effect, and are most useful in combating the paroxysms of the hypertensive disease, angina pectoris; whereas the other members, the nitrites of sodium, erythrol, and mannitose, exert a more prolonged, though a somewhat less energetic, control over persistent arterial tension—lowering the average level of supernormal pressure, and guarding against accessions of it. We should not suppose that every case of supernormal pressure should be treated by some member or other of the nitrite group. To do so would surely remind us of the crude practice of the early days of thermometry, when antipyrin, aconite, and other antifebrile remedies were too frequently prescribed in a routine way to suppress every rise of temperature. The nitrites should, as a rule, be held somewhat in reserve at first, and should not be employed until after the failure or partial failure of the preventive or corrective treatment; and their use and dosage, when indicated, should be the result of experimental observation in each case. The dose and its frequency having thus been properly adjusted, the nitrites may in suitable cases be continued for lengthened periods when necessary; or they may be more particularly prescribed during periods of active increments of pressure which are apt to supervene.

Vaso-dilators may be advantageously combined with cardiac and nerve tonics; but it is always advisable in such cases, every now and then, to watch the effects of the continued use of vaso-dilators on the heart and the nervous system—for all the nitrites tend in the course of time to diminish the tonicity of the ventricle and to impair nerve tone. Should the slightest indications of dilatation appear, they should be combined with small doses of digitalis or strophanthus, or both;[1] and should the nerve tone fail, the vaso-dilators may be associated with small doses of quinine, strychnine, and phosphorus.

Iodine, as potassium and other iodides, is another apressor remedy which has for many years been relied on as our sheet-anchor in the treatment of arterio-sclerosis, whether it is associated or not with a rise in the arterial pressure. And in cases of this type the iodides are often surprisingly well borne in large doses by the mouth, without producing the symptoms of iodism or of depression; but I do not find that the manometer shows that these remedies exert a direct reducing effect on the arterial pressure at all, and therefore my observations agree with those of Stockman, Burnet,[1] Janeway, and some other observers. But notwithstanding this fact, I have often found a long continuance of the iodides in small doses useful in reducing supernormal arterial pressure. I cannot, however, say that I have observed a palpable reduction of the thickening of the accessible arteries from the prolonged use of these remedies, even in full doses. The preparations, iodopin and iodalbin (Parke, Davis & Co.), are perhaps worthy of prolonged trial in arterio-sclerosis.

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[1] A good formula is that of Messrs. Parke, Davis & Co. in their tablets Trinitrin co. (digitalis, strophanthus, and trinitrin). I also find the following pill after meals three times a day useful in hypertension of the arterial wall with slight or moderate supernormal arterial pressure and cardiac dilatation (apex beat displaced to the left with submammary tenderness—a reflex from the overtaxed ventricle): pulv. fol. digitalis, extr. nucis vom. *aa* gr. ⅓, ammon. hippurat., gr. j, pepsin porci (Fairchild), gr. iij, extr. gentian., q.s.

The Amenability of Increased Arterial Pressure to Control.—Cases present decided differences in their response to treatment. This disparity is doubtless largely due to variations in the cause and in the stage of development of the pathological condition. The effects of treatment also differ considerably in the two classes of cases—namely, in the cases in which we can freely prescribe rest and regulation of exercise and life generally, and in those who must follow their avocations and their ordinary routine of life. In the former class the results are often more satisfactory, even when the pressure runs into the higher ranges. But the fact remains that the pressure is much more amenable to control in some cases than in others, in which it may be, and not infrequently is, practically uncontrollable. My observations lead me to the general conclusions—to which, however, I have met with some exceptions—that there is an inverse ratio between the observed reaction to vaso-dilatation and the degree of persistent supernormal pressure; and that in cases following their ordinary course of life the amenability of the pressure to be controlled by treatment diminishes in a general way in proportion to the rise of pressure—the cases having persistent systolic pressures of over 200 mm. Hg. furnishing by far the majority of those which resist appreciable modification by treatment. But these conclusions are only such as might have been anticipated, for a high percentage (probably about 90) of the cases presenting systolic pressure of over 200 mm. are cases of chronic nephritis or of diffuse arterio-sclerosis—with or without renal implication; and in a large proportion of those in which the pressures are below 200 mm. the rise is, as a rule, the outcome of conditions more or less remediable. The percentage of cases regarded as amenable to treatment will also be considerably influenced by the practitioner's conception as to whether the pressure is normal or supernormal in any particular case. There being no such narrow line as that of the normal temperature, each observer, though knowing the general range of the normal pressure, has gradually to build up from his experience a correction for individual instances; being mainly guided in this by the collateral signs and symptoms and by the effects of treatment in borderland cases. When, for example, he discovers that a systolic arterial pressure of 145 or 150 mm., which he regarded as doubtfully normal, may be reduced to, say, 125 or so, with an improvement all round in the patient's condition and health, he will conclude that the latter and not the higher pressure is the patient's normal reading. Such experience is likewise useful in showing that we can thus pick out cases still amenable to treatment which might otherwise be allowed to drift into the less controllable and higher ranges of supernormal pressure. Our clinical management of blood-pressure thus becomes more preventive. It seems to me, therefore, desirable to subject most cases in the doubtful area (from 145 to 160 mm.) to corrective treatment before accepting from 145 to 150 mm. in a routine way as the parting line between the physiological and pathological areas.

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[1] "Therapeutic Action of the Iodides," *The Lancet*, Sept. 8, 1906, p. 646. Dr. Burnet advocates the subcutaneous use of iodopin.

An Increment of the Arterial Pressure is a

Result.—The fact should be emphasised that supernormal arterial pressure is not itself a primary condition to be attacked or controlled as such. It is invariably a result of some pathological cause or causes, and it is, moreover, often indeed a necessary result of conditions which we cannot remove. While realising what are the ill effects of high arterial pressure, and trying to modify them as best we may by our attempts to moderate the pressure, we are apt to forget that that pressure is not infrequently a necessary evil, and should sometimes be regarded as a compensatory effort, or as one of the natural defences of the body. On this point Theodore C. Janeway, who has done so much to advance the clinical study of blood-pressure, has remarked, after referring to the futility of drugs to keep down the blood-pressure: "Another common experience is that the steady downward progress with falling blood-pressure, when heart weakness supervenes, is often made worse by arterial dilators. If improvement comes under digitalis, for instance, the pressure usually rises. These observations find their readiest explanation, if we believe that for such patients an average level of arterial pressure far above the normal is absolutely necessary to ensure the requisite speed of capillary blood flow. For them the optimum pressure for the maintenance of a normal circulation may become 200 mm. or more, instead of 100 to 145 mm. . . . Where no symptoms of a threatening nature exist these preventive measures alone are called for. Under such circumstances a systolic blood-pressure of 200 mm.—yes, even 250 mm. and over—is not incompatible with a number of years of comparative comfort and activity, particularly if the response to treatment is favourable, and the patient co-operates heartily."[1]

ADDENDUM

By Dr. Alfred Mantle

A natural question to ask is, How are we to determine the true position of a case of supernormal pressure? These cases always give us anxiety, and rightly so, for we are confronted with two dangers, and with one eye we see visions of a possible catastrophe through the rupture of a weak vessel by doing too little and with the other the danger of doing too much in lowering the pressure and weakening the cardiac force. My reply to this is, take a careful history of the case, and observe the condition of the cardiovascular, the renal and alimentary systems, and try to find out the cause of the high pressure, such as errors in diet, alcohol, syphilis, lead, and excessive worry, remembering that supernormal pressure is not a disease *per se*, but a symptom only. If the left ventricle is hypertrophied it suggests the pressure has been persistently high for some months or years. Experience teaches us that with a restriction of red meats and alcoholic drinks from the dietary of those who take them freely, the pressure, if not a permanently high one, usually diminishes, and this argues that purine bodies and alcohol probably act as vasoconstrictors. Waste is constantly going on in the body, and it is important to get rid of the products of waste whatever they may be. The proof of the splanchnic vessels being some of the earliest to become affected in arterio-sclerosis points to our relieving congestion of the visceral vessels, which can be done by judicious purgation, and 1 gr. doses of calomel and saline purgatives are most useful two or three times a week. One of the greatest outlets for waste products is the skin, and when inactive a free action of this organ must be encouraged by artificial means, such as by massage, douche and Nauheim baths, when these can be obtained, and intestinal lavation is also very desirable in cases with constipation. It is a common observation that with a dilated periphery causing an active skin we have a lower range of blood-pressure, which leads us to suggest to those who can afford it to follow the sun. The cardinal principles of treatment, then, resolve themselves into a strict regulation of the intake and an active outlet for the waste products, and moderate exercise followed by rest is very desirable to give a better distribution of the blood, but sphygmomanometric observations must be made from time to time as a guidance in treatment.

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[1] "Some Common Misconceptions in the Pathological Physiology of the Circulation and their practical significance," by Theodore C. Janeway, M.D., *New York Medical Journ.*, Feb. 2, 1907.

If, in spite of our regimen and treatment, the pressure continues high, after having watched the case carefully for some months we conclude that this is a case in which the pressure is permanently *supernormal.* In any case we recommend the general regimen to be continued, and so long as no urgent symptoms arise the patient may do his work in life if he avoids excitement and sudden strain.

We must not use any active means to reduce the pressure, remembering that it is probably an advantage and necessary that the pressure should be high for the maintenance of an adequate supply of blood to vital organs. As for drugs of the vaso-dilator type—the nitrite group—do not use them regularly, for it is sounder treatment to get rid of the bodies in the system which contract the periphery by the means already mentioned, rather than by dilating the blood-vessels with the offending poisons still intact.

Iodides should be given very carefully if at all, but if there is any syphilitic history they may be pushed.

A gentleman, aged sixty-five, was referred to me two years ago as having had a high blood-pressure for some years, but he had had a too persistent therapeutic attention paid to it. He had been a great athlete, and rowed in his university boat three years. When I saw him the pressure showed a reading of 180 mm. Hg., and he was dyspnœic on exertion. His heart was enlarged, the apex beat 1½ in. outside the nipple line with very irregular action. There was no albuminuria, and the vessels were not palpably thickened. It was clear that the compensatory hypertrophy which had helped him so long was failing. I gave him no lowering treatment, but that which was directed to the raising of his pressure, advising moderate exercise with plenty of rest between. I gave him strophanthus and nux vomica to raise his pressure, and Nauheim baths and Schott exercises to open out his peripheral circulation which was contracted. After three weeks' treatment his blood-pressure was 15 mm. higher, and the heart more regular, and he returned home much better. He made another visit last year, having taken strophanthus as required, under the direction of his doctor in the interim, and his blood-pressure was in a still more satisfactory condition, for it was steady at 200 mm. Hg., and he was able to do his ordinary amount of walking exercise quite comfortably. This was plainly a case where it was wrong to deprive the patient of a support and force necessary to maintain his smaller circulation, by giving vaso-dilator remedies indefinitely.

It has been one of the unfortunate by-products of our recently acquired ability to determine a patient's blood-pressure in millimetres of mercury that the sight of the column near the top of the tube has so often led to immediate endeavours to reduce the pressure at any cost (Janeway). This point is not sufficiently recognised by some practitioners who make use of the instrument, and I get cases of high blood-pressure sent for special treatment at Harrogate to reduce it, in which nitrites, large doses of iodide of potassium, and other depressing remedies have been used, with the story that the pressure will only partially yield to treatment. The fact is that, owing to the gradual increase of blood-pressure, each individual readjusts his cardio-vascular physiology to compensate this, and the physiological limit is now not 140 or 150 mm. Hg., but may be 220 or 250, and to reduce the pressure below this new physiological limit is not only not indicated, but bad therapy.

The following case is an extreme example of a class not uncommonly met with : High blood-pressure with some cardiac hypertrophy and symptoms for years referable to a certain amount of disturbance of the liver and bowels in the middle-aged, who show no signs of arterio-sclerosis in the systemic periphery, and no clinical evidence of nephritis after examination for albumin and casts. Sir Clifford Allbutt has called attention to these cases, and he names them " hyperpiesis." It is a curable condition if caught early, but if not it frequently ends in a permanently high pressure and possibly in apoplexy or cardiac defeat in trying to overcome the supernormal pressure.

A medical man, aged fifty-eight, was sent six years ago to Harrogate for treatment. He had been seen by two physicians in a northern city who thought the symptoms I am about to describe due to the development of some cerebral disease. The patient had taken a fair amount of alcohol, but none for the five years previous to his illness, but he had been a big eater of red meat. He worked hard in a country practice, and was subject to bad headaches, for which he frequently took calomel and antipyrin. He had, however, what he called a seizure, which he described as being constituted by great pain and throbbing in the head, and marked giddiness in the erect posture. I was asked to see him in consultation some weeks after this attack. He was very emotional, and was rather like a candidate for cerebral softening. He complained of throbbing in the head, and was more or less giddy in the erect posture. On examination, the pulse was one of very high pressure to the touch, and the sphygmomanometer showed a systolic pressure of 220 mm. Hg. As is my custom, I looked for corroboration of this in the position of the heart's apex, which was a full inch outside the nipple line. There was no albumin, and the urine was of good specific gravity. We came to the conclusion that the symptoms were due to the high blood-pressure, and that the only possible chance for the patient was to give him perfect rest with a very strict diet. All meat was forbidden as well as salt. He continued to have nothing but milk and fish for the three or four months whilst under our care. He was well purged and the skin was kept active, but he was much too ill to have any balneological treatment. The blood-pressure gradually came down, and the apex beat came in, and after four months' rest he was able to walk fairly comfortably without much throbbing or pulsation. The patient eventually got back to his work, and has continued well. He lives practically on the same lines of diet, taking no red meat or alcohol, and is now able to do as hard a day's work as he ever did.

CHAPTER IX

THE TREATMENT OF SUBNORMAL ARTERIAL PRESSURE

A Low Arterial Pressure may be Normal to the Individual.—It by no means follows that the arterial pressure is abnormal in any particular case, merely because it is below the average level of pressure generally present in healthy subjects of the same age. I have met with several male adults, even in middle life, in perfect health and enjoying their life work and recreations, in whom the arterial pressure has not exceeded $\frac{95\ \mathrm{S}}{80\ \mathrm{D}}$ mm. Hg. Such subjects are generally slight in build. Then again there is the fact that those in training for games and athletic exercises demanding the expenditure of spurts of energy, have frequently somewhat low arterial pressures during quiescence—the ventricle possessing the potential energy for sustaining the higher arterial pressure required during the stress of vigorous muscular action.

It would therefore seem that a subnormal pulse-pressure, like a slow pulse-rate or a subnormal temperature, may be consistent with good health and the enjoyment of an active life; and is not in itself significant of disease.

A subnormal arterial pressure may, however, be of pathological import when it is conjoined with the signs and symptoms of disease, or impaired health—when, in a word, it is a component of a complex symptom. The pressure normal to the individual may then become actually subnormal. Such pathological depressions are observed in (*a*) some *chronic diseases, e.g.* Addison's disease, anæmia, hæmorrhages, neurasthenia, phthisis and other wasting diseases; and (*b*) in some *acute diseases*, such as diphtheria, typhoid, pneumonia, influenza, and other febrile ailments.

Diet.—The value of meat and meat extractives in the treatment of an asthenic condition of the circulation has of late years been thrown somewhat into the background by our dread of uric acid and the other purine bodies, which are associated with goutiness; and indeed our dietetic directions generally have thus to a considerable extent become vitiated and lop-sided. In low-pressure cases roast meats and gravies should predominate in the diet; and when meats are not admissible in the crude form, as when there is a rise of temperature, extractives of them, which are valuable cardiac stimulants and restoratives, should be given frequently in small quantities. In many cases of acute illness (such as diphtheria, pneumonia, influenza) in which the heart is apt to fail, watery extracts of muscle, when given in judicious intervals, often afford timely aid in tiding the heart over a critical period, acting, like other stimulant remedies, quickly and decisively, and enforcing the sustaining effects of food (milk, etc.) which requires time for its absorption and assimilation. They should not be given, as they were some years ago, as substitutes for food. Alcohol (especially in the form of brandy egg-flip) acts in a similar way when administered with food. Sodium chloride is another stimulant of the circulation which is apt to be overlooked. It should never be omitted from the liquid foods, such as milk; and I would suggest that it should be given along with calcium and potassium chloride in the proportions present in Ringer's circulating fluid,[1] which may form the menstruum of the drinks—lemon, barley or toast water.

The due apportionment of rest and the regulation of exercise are most important in the treatment of low-pressure patients. In acute cases absolute recumbency is of course necessary. In chronic cases and during convalescence from febrile ailments, the prescription of exercise frequently requires to be well guarded, to prevent recurrence of atonia and consequent delay in recovery; and in such cases recumbency should be enjoined during the hypotonic periods following the meals and exercise, when undue draining of blood into the splanchnic veins takes place. In low-pressure cases abdominal massage and faradism of the abdomen are useful.

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[1] Aseptules containing the chlorides of sodium (6 parts), potassium (3 parts), and calcium (1 part) are supplied by Messrs. Oppenheimer, Son & Co. for the ready preparation of Ringer's solution—one aseptule being dissolved in 20 oz. of freshly boiled water.

The medicinal treatment of subnormal pressure by cardiac and vaso-motor tonics is so well known that more than a passing reference to it is unnecessary. Strychnine, digitalis,[1] and adrenaline are the best remedies of this class; and phosphorus, arsenic, and iron are frequently useful adjuncts. As a pressor remedy formic acid, according to Clément[2] of Lyons, is promising; this observer having shown that it is a powerful tonic of muscle fibre—whether striped or unstriped—and quickly abolishes the sense of fatigue. The tonic effects of formic acid or the formates is much appreciated by French physicians.

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[1] The value of digitalin in the low arterial pressure of Addison's disease is well shown by Rendle Short, *The Lancet*, vol. ii, 1906, p. 285.

[2] *L'Acide Formique et la Force Musculaire*, par le Docteur Clément de Lyon: Paris, 1905.

The Depressor Effect of the Toxins of Diphtheria and of other Infective Febrile Diseases may be met by the Routine Administration of Strychnine. —This fact in regard to diphtheria is thus clearly stated by James Davidson: "As practically all the cases of diphtheria in the Edinburgh City Hospital were by routine practice put upon strychnine and alcohol on admission, the fall of blood-pressure due to the toxic condition was to a large extent annulled. In cases which did not receive any such stimulant a fall was in every case noted until this treatment was commenced. During the routine administration of such stimulants over long periods, the blood-pressure response to any single dose was apparently absent, or in any case was much less marked than that following the first few doses given to the patient; but in these circumstances, if the routine order were suspended for one or two periods, the blood-pressure was noticed to be affected. This constant blood-pressure level, as maintained by the use of strychnine at regular intervals, seems to represent the ideal to be aimed at in the stimulant treatment of toxic conditions such as diphtheria."[1]

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[1] "Blood-Pressure in Fevers," by James Davidson, M.D., etc., *The Lancet*, vol. ii, 1907, p. 1087.

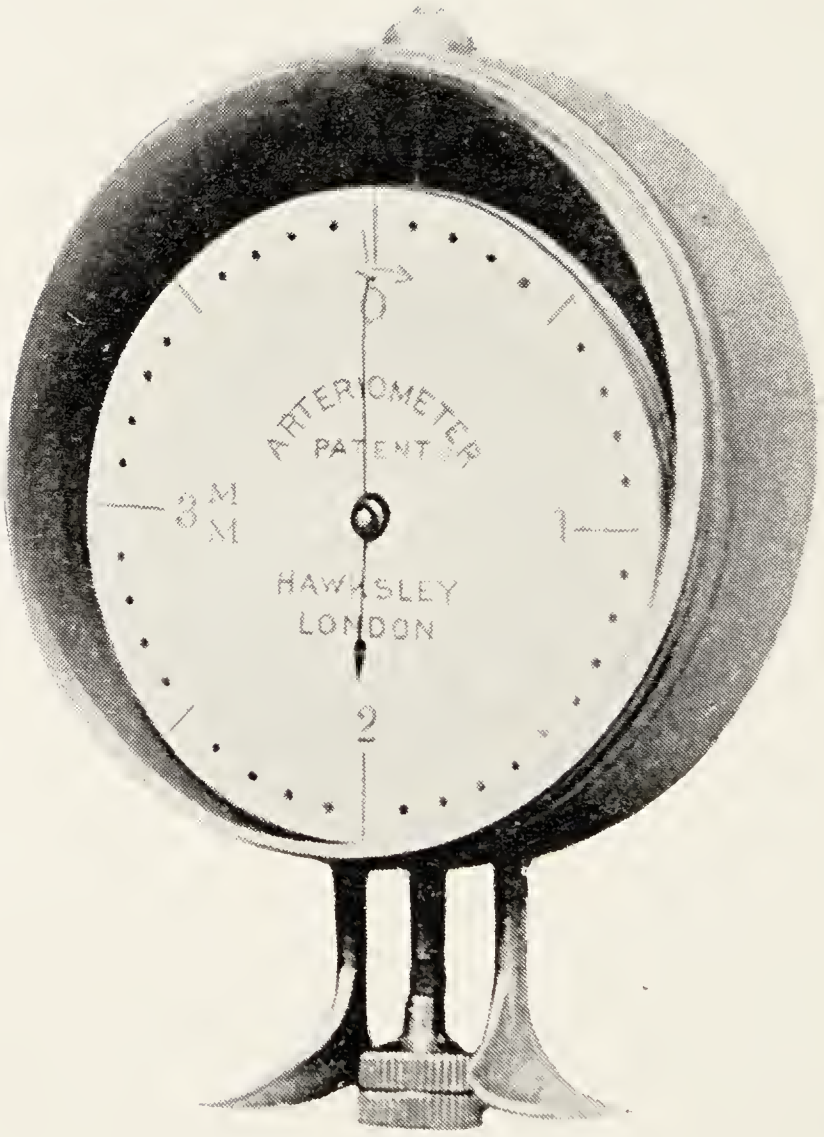

FIG. 6.—THE ARTERIOMETER. (Full-size.)

The arteriometer is made by Mr. Hawksley, 357 Oxford Street, London, W.

To face page 209.

CHAPTER X

ON ARTERIOMETRY

HAVING found the calibration of the radial artery useful and instructive when made by the side of blood-pressure measurements, I will describe the little instrument (Fig. 6) which I devised some years ago for the purpose of determining the internal diameter or calibre of the artery. It is applied in the vertical position, *i.e.* at right angles to the artery and to the arm (see Fig. 7). Each observation comprises two stages. In the first the pointer and the dial travel for a certain distance, which varies in each case, from *right to left*—then the dial ceases to move. In the second stage the pointer suddenly leaves the stationary zero and travels from *left to right*—in the direction of the arrow on the dial. During this second stage the artery is being closed, and the excursion of the pointer from zero to the point at which pulsation beyond the pad completely ceases provides a measure of the calibre of the vessel.

I. How to use the Arteriometer

The Wrist-rest.—The box in which the instrument is carried, when opened and inverted, becomes a convenient wrist-rest. The strap which surrounds it provides three variations of extension, any one of which may be selected as the best adapted to the individual extensibility of the wrist, which varies in different cases.

Directions.—1. Extend the wrist (preferably the right one) over the wrist-rest, and see that the elbow leans on the table and that all the muscles are relaxed; raise slightly the radial side;[1] put the left hand over the upturned palm and thumb, and thus maintain the contact of the dorsum against the wrist-rest and prevent the thumb from being extended; and place the pad of the left index finger over the radial artery, so as to close the vessel and exclude a reflux beat that may be present, when the tip only will perceive the direct pulsation. The artery and the adjacent structures are thus rendered slightly tense and at rest—conditions which are necessary to definite observation.

2. Examine the bed of the artery with the finger

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[1] This direction is of special importance in taking the radial calibre in the recumbent posture; for then the patient is very apt to evert the supine forearm.

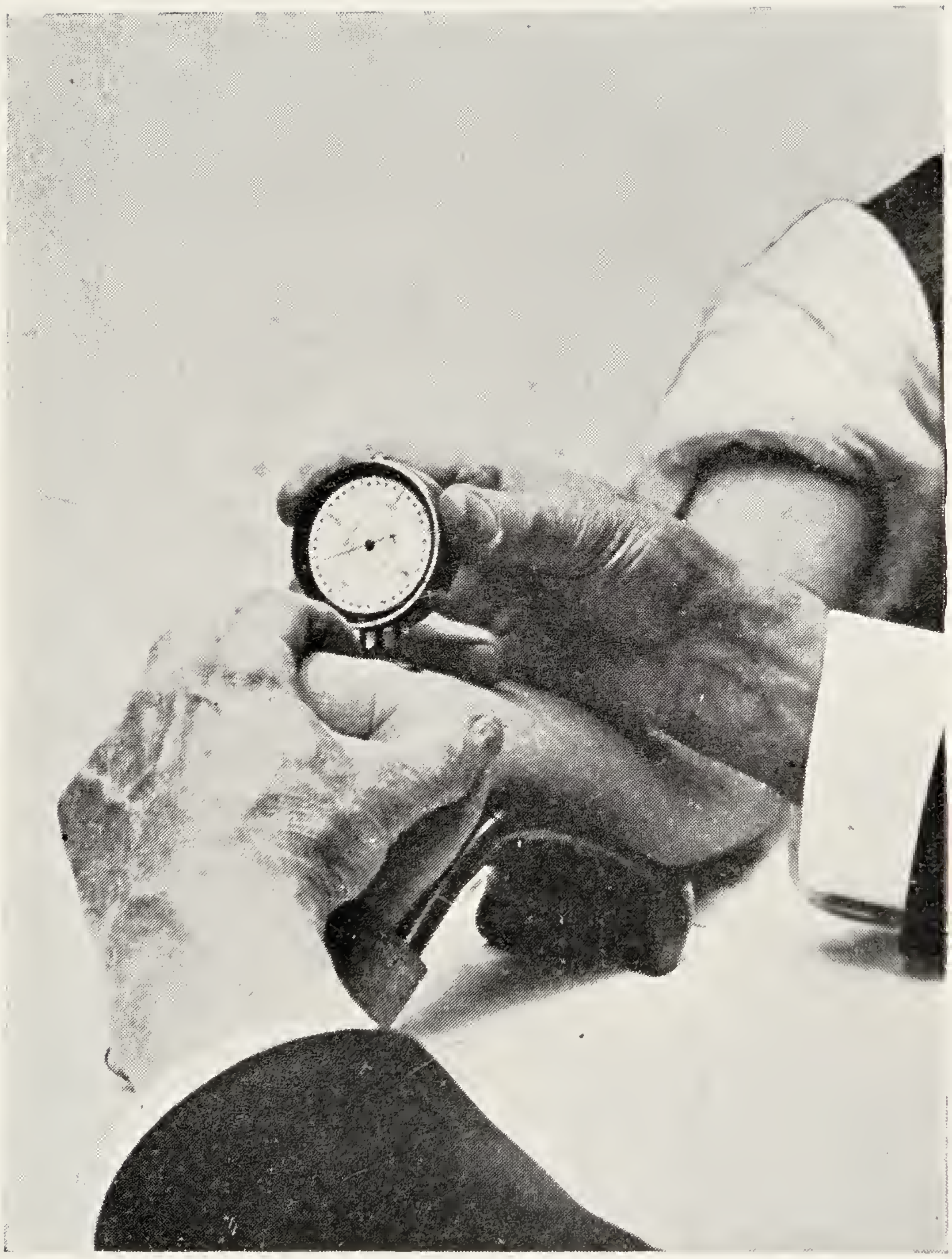

FIG. 7.—MODE OF USING THE ARTERIOMETER, THE WRIST BEING EXTENDED ON A WRIST-REST.

To face page 210.

or the blunt end of a pencil, test the closing of it, and select the best site for the observation. If this should not happen to be in a line with the prominent part of the styloid process, which forms a good general landmark for this observation, stain the site with a dermographic pencil.

3. Hold the arteriometer between the index finger and the thumb of the right hand, and plant the foot-rests, one on the styloid process and the other on the flexor tendons, rendered tense by the extension of the wrist, and adjust the pad carefully over the site of the artery. It is important that the observer should see that the inner foot-rest is well supported by the tendons, and if these are not felt to be firm enough the wrist should be somewhat further extended. The instrument should be held, throughout the observation, perpendicularly to the plane of the limb ; and *should not be inclined backwards* (see Fig. 7).

4. On applying the instrument, it will be observed that the indicator travels on the dial in the direction opposite to that of the arrow, and generally for more than one revolution, and in doing so, on reaching zero on the index, it rotates the dial. The extent of this excursion to the left, which takes place before the radial measurement can be made, depends on the relation which the site of the artery

bears to the two fixed points on which the foot-rests are planted, *e.g.* whereas in the average formation it may not exceed a revolution and a half, it may, when the wrist is round and prominent, approach or complete two revolutions, and when exceptionally shallow, it may not compass more than one. The mechanism allows of two backward revolutions. The stem may be shortened or lengthened by screwing the pad up or down. When it occupies the medium position, indicated by a line across the middle of the flat portion of the screw, on applying the instrument, the indicator settles in almost every case in some part of the second revolution.[1] The instrument should be applied somewhat gradually, so as to avoid propelling the zero point of the dial in advance of the pointer.

The observation proceeds by slowly pushing down the outer case—care being taken to bear equally on both foot-rests—when the indicator will recede to the left a few points farther, and then will *suddenly move in the opposite direction*—that of the arrow—leaving the dial stationary.

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[1] In quite exceptional instances the pointer may barely complete one revolution before it leaves zero—when the pad should be lengthened by unscrewing it; or it may actually complete the second revolution—when the pad should be shortened by screwing it up a little.

This point indicates *the commencement of the radial measurement*; and, in order to make it as definite as possible, it is necessary carefully to preserve the same line of pressure. It is advisable to repeat the turning point of the movement of the indicator a few times by raising and lowering the case, which is then pushed farther downwards, until the artery is gradually closed, when pulsation is no longer perceived by the tip of the index finger.[1] The completion of the observation should be quite definite, and this may be repeatedly tested by allowing the pulsation to return, on slightly relaxing the downward pressure on the case, so that the indicator may recede a point or two, and then to obliterate it as before.[2] *The interval traversed by the indicator, from the point when it leaves zero and moves in the direction of the arrow to that which shows the artery to be occluded, affords the measurement of the calibre.* If, however, pulsation does not cease after the indicator has measured off as much as 3·0 mm., the observer should suspect that either the pad is incorrectly placed in regard to the artery, or the line of downward movement of the pad has not been made at right angles to the resistant plane on which the artery rests, or the spot chosen has not been sufficiently adapted to closure of the vessel from the want of counter-support, etc., or perhaps the structural conditions are altogether unsuitable for this mode of observation.

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[1] When the "reflux" or "recurrent" beat from the palmar arch is present, it is excluded by the pad of the finger.

[2] It will be found that in most cases the well-trained finger does not require more than $\frac{1}{20}$ of a mm. for the purpose of defining the absence of pulsation.

Normal Postural Variation.—The calibre follows the variations of the arterial pressure due to gravity and change of posture. In assuming the erect postures (sitting or standing) it immediately attains its maximum, and just as quickly falls to its minimum in recumbency. As a rule, in healthy subjects the postural variation extends from ·4 to ·6 mm., the average calibre in the erect postures being from 2·0 to 2·3 mm. and in recumbency 1·5 to 1·8 mm.

II. Epitome of Clinical Observation of the Radial Calibre

In the clinical field the arteriometer affords useful information concerning disturbances of the vaso-motor system and the state of the arterial wall. It therefore determines important facts relating to the circulatory mechanism; whereas the manometer merely measures the blood-pressure as distinct from the condition of the arteries. The two instruments are therefore complementary to each other in clinical research bearing on the circulation.

Reversed Postural Variation.—When the arterial pressure is lowered by a reduction of tone in the arterial wall, inducing splanchnic stasis, the postural measurements of the radial calibre are reversed—becoming maximum in recumbency and minimum in the erect postures. In this way the arteriometer enables the observer to measure the effects of fatigue on the circulatory system in whatever way induced, whether by exercise, heat, ill-health (acute or chronic), hæmorrhage, or otherwise.

The Radial Calibre is Subnormal.—This may indicate: (*a*) A congenitally small radial artery merely, when the arterial pressure and the postural variations of the calibre will be normal. (*b*) A subnormal arterial pressure, with general wasting of tissue and reduction of the volume of the blood. (*c*) Arterial constriction with raised arterial pressure and an absolute or relative reduction of the venous pressure, the postural variation of the calibre ceasing should the constriction be very persistent and decided. This condition of the arteries is observed in the early stage of arterio-sclerosis—the stage which has been termed pre-sclerotic.

(*d*) Endarteritis (from syphilis, lead, gout, etc.), atheroma, and arterio-sclerosis. The calibre is not only reduced, but has lost its postural variation.

The Radial Calibre is Supernormal.—This may indicate: (*a*) A normally large radial artery, when the arterial pressure and the postural variation of the calibre will be normal. (*b*) An excessive arterial blood-pressure overcoming the arterial tonus.

The Arteriometer is Useful in suggesting Vaso-dilator or Vaso-contractor Remedies and in measuring their Effects.—I have frequently found the arteriometer of considerable service in indicating the line of treatment to be followed; suggesting the adoption of measures and remedies which either reduce a hypertensive or which raise a hypotensive state of the arterial walls. The conjoint use of it and the manometer is a source of sustained interest in enabling one to follow the relation between the vaso-motor state of the arteries and the blood-pressure, and thus daily to exemplify in practice our physiological knowledge of that relation.

CHAPTER XI

ON VENOUS AND CAPILLARY PRESSURE

The Venous Pressure may be determined by ascertaining the Pressure required to permit Refilling of a Collapsed Vein.—Select a vein free from branches on the dorsum of the hand, the wrist, or the forearm. It should possess an efficient valve—a fact proved by placing a finger on its distal end and emptying the vein with another finger drawn along it, when the piece of vein as far as the valve will remain collapsed. Place the phalangeal pad across the distal end of the vein, and adjust the strap around the hand, wrist, or forearm without compression, and buckle. Inflate the bag to the pressure of 40 mm. and empty the vein. Then *very gradually* release the pressure, when at a certain point in the fall, which denotes the venous pressure, the vein suddenly refills.

The Venous Pressure may also be determined by Gravity.—I have observed that the venous pressure may be accurately measured without the aid of a blood-pressure instrument; a foot-rule or measuring-tape being all that is necessary. If the veins on the dorsum of the hand are sufficiently visible, it is found that when the hand is held in the vertical position, with the fingers extended, and is very gradually raised, the veins, at a certain height above the level of the apex of the heart, are seen to collapse quite suddenly. The completion of the observation is definite—for the transition from a partial to a complete collapse takes place within the space of half an inch. It is observed that the veins nearest the fingers collapse somewhat sooner than those nearest the wrist; but the difference is so slight that I prefer to take the collapse of all the veins of the dorsum as the indication for measurement.

I conclude that at the moment of collapse of the veins the blood-pressure within them is practically nil—being balanced, as it were, by the force of gravity; and that we may express this point of annulling the hydrostatic rise of the blood in mm. Hg. pressure. This may be done by making a very simple calculation. If we take the average specific gravity of the blood as 1·060, and that of mercury as 13·570, the 25·5 mm. contained in one inch will represent 1·985 mm. Hg. (or approximately 2 mm. Hg.). Therefore, if we multiply by two the number of inches above the level of the apex of the heart at which the veins collapse, we ascertain in mm. Hg. the venous pressure.

This simple method of measuring the venous pressure has afforded me more uniform results than other methods; and, as it enables one to discriminate between differences of 1 mm., it is definite and delicate. In applying it, it is important to see that the pressure is not artifically raised. This may occur through nervous perturbation, or obstruction of the venous flow by tight clothing, or even by the observer placing his hand round the forearm. The lowering effect of cold on the venous pressure should also be kept in mind.

I am disposed to think that this gravity method yields an approximate reading of the capillary pressure; for, as a rule, just when the vein collapses there is observed a slight blanching of the nail—especially its central portion—which is made somewhat more apparent when the nails of the opposite hand, with the fingers flexed downwards—so as to retain their blood in the elevated position—are compared with the uplifted nails side by side. The measurement from the apex of the heart to the nails gives, as a rule, a reading for the capillary pressure 8 or 10 mm. Hg. higher than that of the venous pressure—*e.g.* capillary pressure, 25–30; venous pressure, 15–20 mm. Hg.

The methods of observing the venous pressure on the dorsum of the hand have yielded the following results:

Posture.—The pressure in normal subjects is lower in the recumbent than in the erect postion, and intermediate in the sitting posture: *e.g.* recumbency 10–15, sitting without support 15–20, and standing 20–25 mm. Hg.

Muscular Action has a pronounced effect in raising the venous pressure. This is even observed in the postures; for example, it may be 10–15 mm. Hg. while sitting with back supported and all the muscles relaxed, and on sitting erect it may rise to 15–20 mm. Hg. But the increment is much more decided in active exercise of all kinds; even in cycling on a level road (and therefore with only slight effort) the venous pressure may be doubled —*e.g.* 15–20, rising to over 30–40 mm. Hg.; and in cycling on a rising gradient it may exceed 50 mm. Hg.

Contraction of the muscles, by compressing the veins, raises the pressure: this fact, which was utilised in the days of venesection, is well shown by raising the hand until the veins collapse, when muscular contraction of the arm refills them.

Mental Exercise and Psychological Excitement and Perturbation likewise increase the pressure. Hence the importance of quiescence of mind and absence of emotion during the observation.

Digestion also raises the venous pressure. Its augmenting influence is, however, counteracted by the reducing effect of the splanchnic drain which takes place after a meal—especially during the first hour. But, notwithstanding this fact, the systemic venous pressure does rise as a rule even in the erect postures during digestion.

Temperature.—Warmth raises the venous pressure and cold lowers it.

Respiration influences the pressure quickly and powerfully. All forms of effort which increase the intra-thoracic air-pressure raise the venous pressure markedly ; such as coughing, straining at stool, lifting a heavy weight with the glottis closed, Valsalva's experiment (forcible expiration with closed mouth and nostrils).

The effects of deep inspiration and expiration on the venous pressure are readily studied by gradually raising the extended hand until the veins on the dorsum *just* collapse (*see* p. 218), maintaining it at that level and taking a series of long-drawn, deep inspirations and prolonged expirations, when the veins will be seen to fill with every inspiration and to empty with every expiration. These respiratory variations in the collapsed veins show the far-reaching effect of deep breathing on the peripheral circulation—the rise and fall in the venous pressure being indicative of the same variations in the capillary and tissue lymph circulation. These observations are not, however, quite in keeping with what we know of the dilating effect of inspiration and the contracting effect of expiration on the intra-pulmonary vessels, which should induce a fall in the venous pressure during inspiration and a rise of it during expiration. But this discrepancy may be accounted for by (1) the fact that the variations in blood-pressure produced by inspiration and expiration are actually not synchronous with the respiratory movements —the maximum effect of each being intermediate between inspiration and expiration;[1] and by (2) the muscular action on which deep and prolonged inspiration entirely depends—expiration being nearly independent of it.

Sleep and drowsiness, by reason of the vasomotor relaxation induced, favour a rise in venous pressure.

How is the Venous Pressure modified?—The following are the leading factors: in *reduction*—(1) diminution of the ventricular output and contractility, (2) contraction of the arterioles, and (3) splanchnic drain; in *increase*—(1) augmentation of the ventricular output and contractility, (2) vaso-dilatation, (3) increased intra-thoracic air-pressure, and (4) compression of veins during muscular contraction.

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[1] *A Manual of Physiology,* by G. N. Stewart, M.D., etc., London, 1899, p. 249.

The Venous Pressure as an Indicator of the State of the Peripheral Circulation.—This physiological study of the venous pressure shows that a rise of the venous pressure is an external and visible sign of increased activity in the peripheral circulation—indicating the passage of a larger volume of blood through the capillary area; and a fall in that pressure shows either diversion of blood to the spanchnic area or a contraction of the peripheral vessels.

The Relation between the Venous and the Arterial Pressures.—The arterial as well as the venous pressure is raised during active exercise and during muscular contraction and effort; but in quiescent conditions of the body, when the venous pressure is raised by either warmth, sleep or digestion, the proximal arterial pressure falls, while the distal arterial pressure rises as part of the increment of peripheral blood-pressure.

The Capillary Blood-pressure

The Agreement between the Variations of the Venous and Capillary Blood-pressure.—Observation has shown me that variations of the capillary pressure are more definitely afforded by the venous pressure than by the methods of direct measurement which have been proposed. I have found that the venous pressure taken by the gravity method is the best indicator of the capillary pressure, when the hand is normally warm; this is an important proviso, for when the hand is cold vaso-contraction of the venules takes place, which favour a rise in the capillary pressure, while it determines a fall in the venous pressure.

The Amount of Tissue-fluid indicates the Capillary Pressure.—But observation has further demonstrated that the amount of tissue-fluid present in the last phalanx is the best and most trustworthy guide to the variations of the capillary pressure. The method of observation of the tissue-fluid is described in the Appendix.

The Relation between the Venous Pressure and the Tissue-fluid.—As the venous pressure is a good gauge of the capillary pressure, there should be a similar relationship between its variations and those of the volume of lymph which circulates through the interstitial tissue. Observation shows that this is so. I have found that (1) whenever the veins on the dorsum of the hand collapse at the level of the cardiac area (from apex to base) the reading of the tissue-fluid falls to zero (*i.e.* the lymphometer reading indicates either an absence of tissue-fluid or the presence of merely a point or two of lymph); and (2) the amount of lymph rises with the increment of venous pressure, as shown by the veins having a higher collapsing point than the cardiac area.

These and other observations have shown (1) that the tissue-lymph circulation in the systemic area is but an extension of the capillary circulation, and (2) that it is tidal, or to and fro in character—a movement which is the outcome of the rise and fall in the capillary blood-pressure.

APPENDIX

THE HÆMACYTOMETER, LYMPHOMETER, AND HÆMOGLOBINOMETER

I. The Hæmacytometer

The Principle followed.—The method is based on the following facts; (*a*) When a candle-flame is viewed through a glass tube containing water, a transverse line of bright illumination is seen, consisting of closely packed, minute images of the flame produced by the longitudinal fibrillation of the glass. In the process of drawing out, the tube becomes minutely corrugated or fibrillated in the direction of its axis. The corrugations are extremely small, but they are easily observed when a tube is held before a flame and the structure of the glass is examined by a pocket lens, when the corrugations are seen to produce alternate bright and shaded lines disposed in the vertical direction. Each corrugation acts as a lens, hence the horizontal line made up of a congeries of images of the flame. (*b*) When some fixing fluid (such as Hayem's) is added to blood, the mixture presents a certain degree of opacity according to the amount of blood or of the fixing fluid. (*c*) The opacity thus produced completely shuts out of view the illuminated line, until, on diluting further, a definite point is reached, when it can just be detected as a streak of light *across* the tube—the dawn,

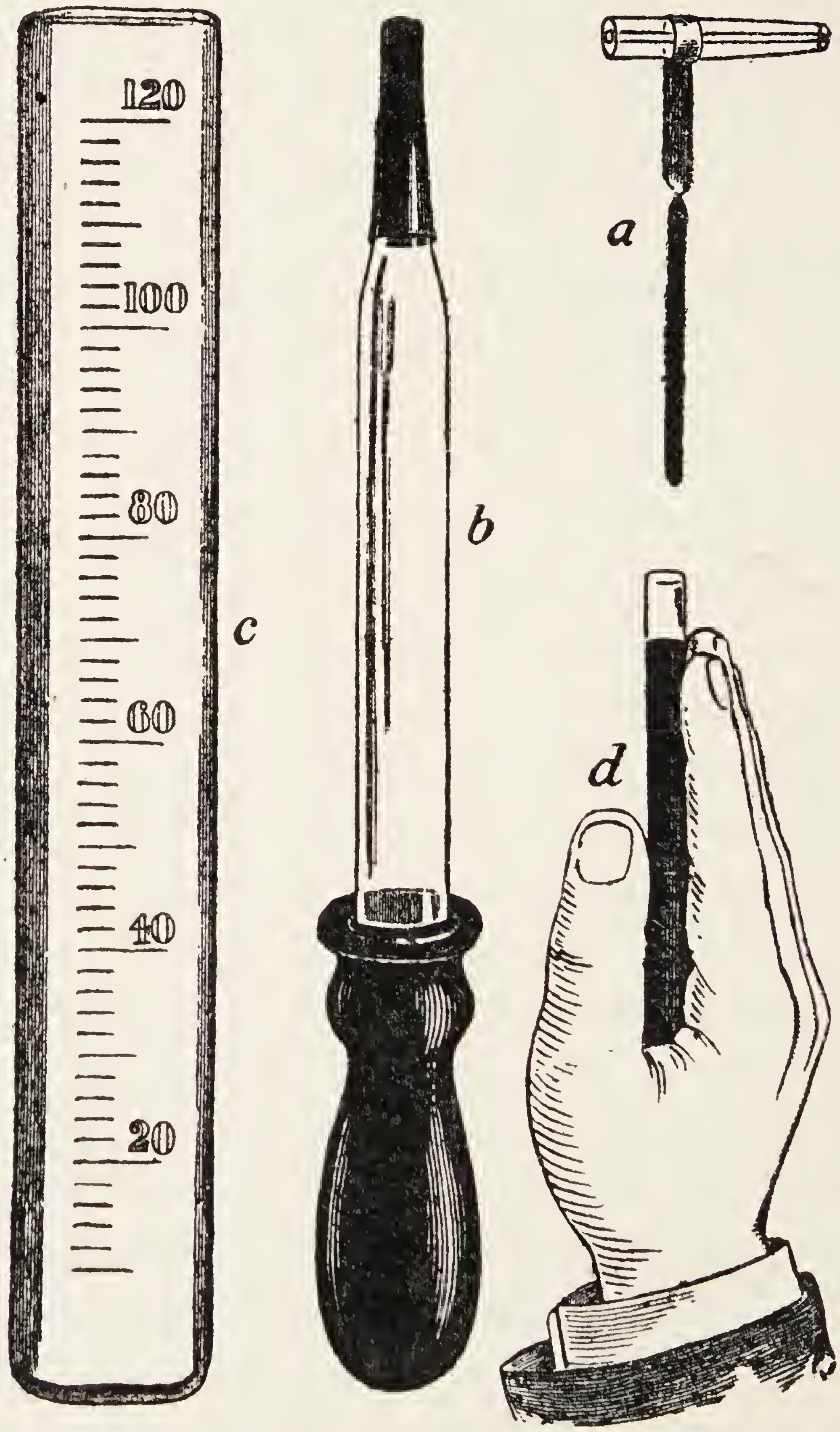

FIG. 8.—THE HÆMACYTOMETER.

a, Blood measurer; *b*, mixing pipette; *c*, flattened graduated tube; *d*, mode of holding the tube for observation.

as it were, of the bright minute images of the flame seen when the opacity is absent or is further reduced.

Repeated observation has shown that the development of this delicate horizontal line by dilution with the fixing fluid is a very sensitive indicator of the percentage of the blood corpuscles.

The apparatus consists of (1) a graduated flattened tube ; (2) an automatic blood measurer ; (3) a mixing pipette ; (4) a candle ; and (5) a bottle of Hayem's solution [1] (see Fig. 8).

The determination of the scale was made by the enumeration method extended to 120 squares applied to the blood of normal man. One hundred degrees presents the generally accepted standard of 5,000,000 corpuscles per mm. ; therefore 80 degrees is equivalent to 4,000,000, etc.

The white corpuscles in a state of health do not affect the readings.

The Mode of Observation.—The pipette, previously dried by passing through it a needle threaded with cotton, having been accurately filled and cleared from adherent blood by the finger, the rubber nozzle of the mixer filled with Hayem's solution is then applied over its bevelled end, and the blood is thoroughly washed into the flattened tube (Fig. 9). When the blood is not suspected to be particularly poor in hæmoglobin and corpuscles, the amount of fixing fluid added in the first instance may raise the column to the 60 or 70 per cent. mark ; otherwise the first charge should be less. The contents of the tube are then uniformly mixed by inverting the tube a few times with the thumb over its mouth, care being taken on removing the thumb to draw it over the lip of the tube so as to restore as much as possible the fluid adhering to it. This procedure should of course be repeated after every addition of the diluting fluid. The observation is made in a dark room or in a darkened room free from cross-lights. The observer stands ten feet from the flame. In making the observation it is important to shut out as much as possible the diffused light of the candle. The most satisfactory way to do this is to place the lower end of the tube, with its long diameter

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[1] The formula is as follows: Hydrargyri perchloridum, gramme 0·5 ; sodii chlorid., gramme 1·0 ; sodæ sulphas, grammes 5·0 ; aqua distillata, 200 c.c.

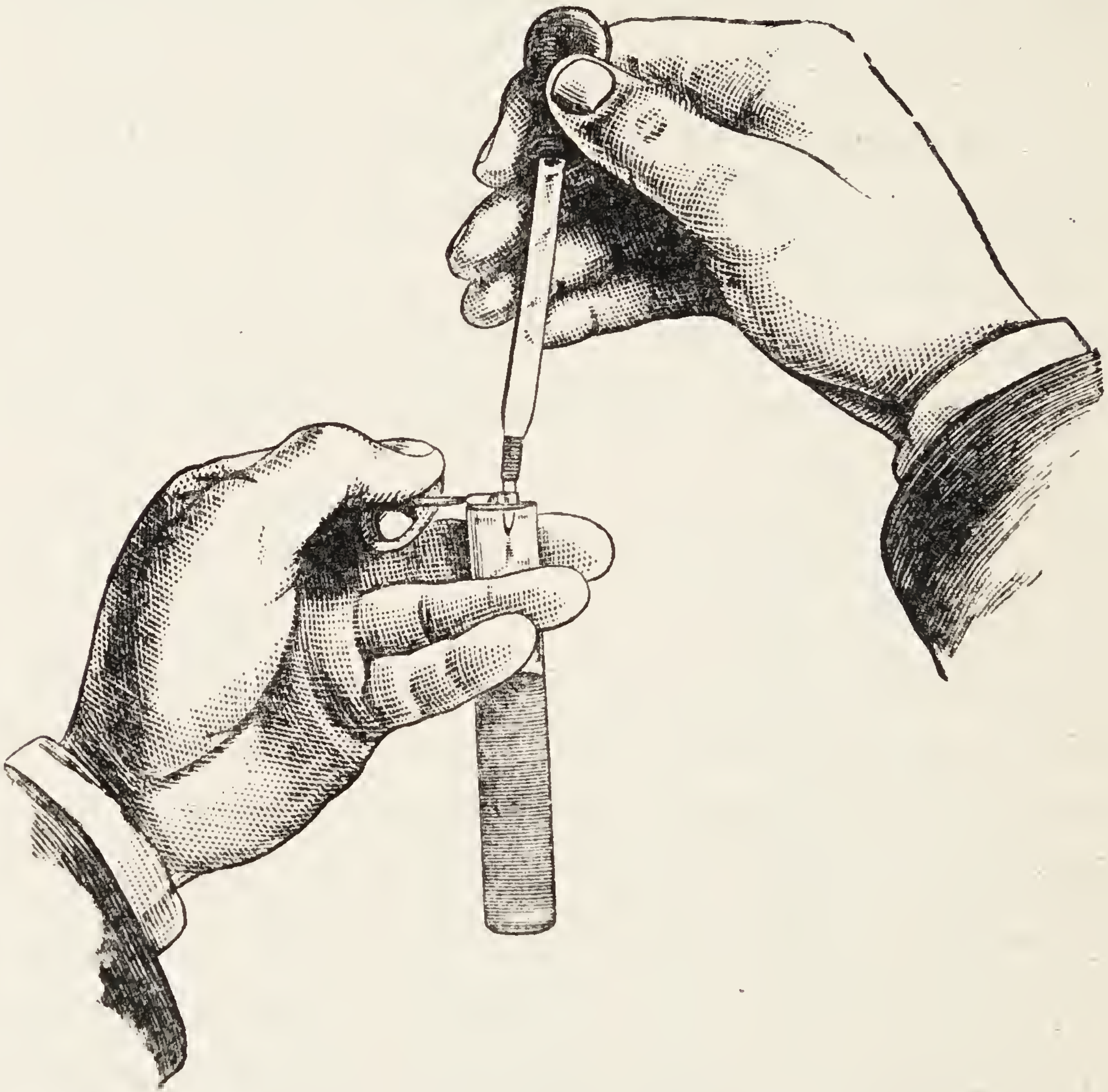

FIG. 9.—WASHING THE BLOOD INTO THE HÆMACYTOMETER TUBE WITH HAYEM'S SOLUTION.

in a line with the candle, in the concavity between thumb and forefinger, so as to make as it were a frame for it, and then to bring this part of the tube *quite close* to the eye (Fig. 8,*d*). The observer must look out sharply for the first appearance of the faint transverse line of light which dilution brings into view, and the earliest indications are obtained by turning the tube on its axis, when the line will first become visible at the sides of the tube.

II. The Lymphometer

The hæmacytometer, adapted to the measurement of tissue-lymph, consists of the following apparatus :

(1) Set of lancet prickers.
(2) Two measuring pipettes.
(3) Two hæmacytometer tubes.
(4) A set of stout rubber rings.
(5) A fimbriated tube.
(6) A nipple pipette.

The method of observation [1] was described in a preliminary note "On the measurement of tissue fluid" read before the Royal Society, June 11, 1903. It is based on an examination of two samples of blood ; one before, and the other after compression of the tissues of the finger by stout rubber rings. The first sample is believed to contain a certain admixture of tissue fluid ; for the needle, in puncturing the capillaries, must penetrate the areolar tissue spaces which surround them, and perhaps also the lymphatic capillaries of origin under the epithelium,[1] and so must liberate lymph, which mingles with the blood. On the other hand, it has been concluded that the second sample is freed from that admixture, as, after removing the rings which squeeze the lymph away from the capillaries, the blood instantly returns to the vessels, whereas an appreciable interval elapses before fresh lymph is formed.

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[1] See the Oliver-Sharpey Lectures, *The Lancet*, vol. i, 1904.

A puncture is made on the dorsum of the finger near the root of the nail—this part being much more amenable to efficient compression than the pad of the finger—and the blood is measured off by one of the automatic pipettes; after which the three rings are slowly rolled with pressure over the finger and are lodged beyond the phalangeal joint. The metal tube is then passed over the finger up to the joint, and the rings are rolled on to it, and are thus removed without compressing the distal parts of the finger. Now the blanched finger suddenly reddens, when (the lymph having been squeezed away by the rings) the blood may be extruded by pressure through the original puncture without fear of disturbing its quality. It is then measured off by the other pipette, and each sample is washed into its respective tube, and the percentages of corpuscles are read in the manner previously described (see p. 229). This method of enumeration enables the finest differences between the two samples to be discriminated. When tissue-lymph is present the percentage quantity is indicated by the higher reading of the tube containing the sample after compression; but when it is not present the tubes read alike. A nominal difference of two points may be regarded as practically indicating a minimum quantity of tissue fluid.

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[1] "The Lymphatics," by G. Delamere, P. Poirier, and B. Ciméo; translated by Cecil H. Leaf, M.A., M.B., F.R.C.S. (Eng.), 1903, p. 3.

Besides affording a measure of the tissue-fluid in the finger, I have found this method useful in giving a ready all-round estimate of the condition of the blood, by taking the mean of the two observations, *e.g.*—

92 after compression,
80 before ,, = 86.

These observations occupy but a few minutes; and the practical advantages derived from them are considerable in at once settling the question of anæmia (in its broad sense), spanæmia and chlorosis; and in detecting polycythæmic or apoplasmic anæmia in which the blood is concentrated and therefore reduced in volume (see "Blood and Blood-pressure," by G. Oliver, 1901, pp. 98–102).

III. The Hæmoglobinometer

The method consists in comparing a specimen of blood suitably diluted with water in a shallow white palette with a number of standard tests very carefully prepared by the use of Lovibond's coloured glasses. The capillary pipette *c* (Fig. 10) is first filled with blood obtained by pricking the finger. This is washed

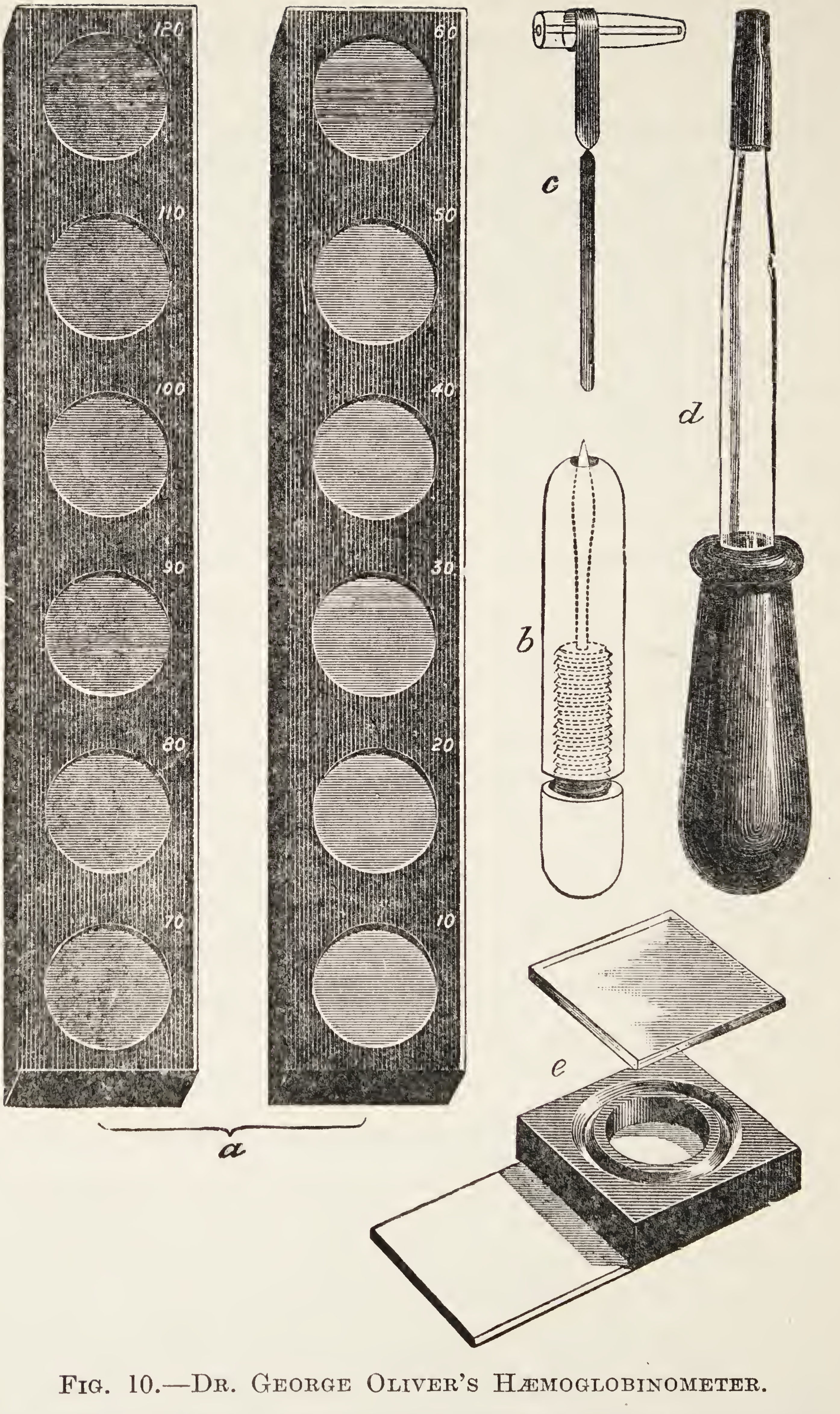

FIG. 10.—DR. GEORGE OLIVER'S HÆMOGLOBINOMETER.

a, Standard gradations; *b*, lancet; *c*, capillary measuring pipette; *d*, mixing pipette; *e*, blood cell and cover glass.

with water by the mixing pipette *d* into the blood-cell, *e*; the cell is then just filled with water, and the blood and water thoroughly mixed by the handle of *c* being used as a stirrer. The cover glass is then adjusted, when a small bubble should form, a clear sign that the cell has not been overfilled. The cell is then placed by the side of the standard gradations, and the eye quickly recognises its approximate position on the scale. The camera tube provided with the instrument will more accurately define it. Artificial light should be used. If it is proved that the blood solution is matched in depth of colour by one of the standard grades the observation is at an end; but if the tint is higher than one grade, but lower than another, the blood cell is placed opposite to the former, and riders (not shown in the illustration) are added until the match is accurate. The standard gradations are marked in percentages; thus 100 per cent. represents the normal (corresponding to 18·5 c.c. of oxygen per 100 c.c. of blood); if, for instance, the blood is so poor that it only contains half the normal quantity of oxyhæmoglobin, the blood sample diluted as described will be matched by the grade marked 50, and so for other percentages.[1]

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[1] These instruments are made by Mr. Lovibond, the Colour Laboratories, Salisbury.

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